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THE BROWN ROT DISEASES OF FRUIT TREES

By

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FOREWORD

No one knows exactly how much damage is caused annually by the Brown Rot diseases of fruit and fruit trees, but the figure is certainly high and has mounted appreciably in recent years. These diseases are world-wide in their occurrence, and a vast amount of research has been carried out on them in many countries. Yet despite these efforts, no really effective method has been found of keeping them under control.

The time is ripe to take stock of the situation, and the Ministry is indebted to Dr. Wormald for undertaking the difficult task of bringing together in the form of a monograph all that is known about these diseases and the fungi that cause them. He is well qualified to do so. It is nearly forty years since he himself laid the foundations of our knowledge of Brown Rot diseases, in the thorough and authoritative manner which has characterized all his many contributions to the study of fruit tree pathology during a long and distinguished career at Wye College and the East Malling Research Station. The bulletin is written primarily for the plant pathologist and technical specialist, and is therefore appropriately included in the Technical Series; but there is much of direct interest in it for practical growers.

An earlier publication by Dr. Wormald, covering our knowledge of the Brown Rot diseases up to 1934, was published by the Ministry in the following year, as Bulletin 88.

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January, 1954.

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H. WORMALD

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THE BROWN ROT DISEASES OF FRUIT TREES

The term "Brown Rot" refers primarily to the discoloration and decay of maturing fruit as instanced by the common rot of apples, a disease that cannot fail to be observed by all fruit-growers and gardeners at the time the fruit is ripening or is being gathered; many fallen fruits, too, show the rot at various stages of development. A similar rot occurs on pears and on the stone fruits, plums, cherries, peaches and apricots. Fruit decay, showing as a brown discoloration of the flesh, may be produced by any one of a variety of parasitic fungi, or it may be the result of a non-parasitic physiological "breakdown". But the term "Brown Rot", in its technical and restricted sense, is used to denote the type of decay brought about by one or other of three closely related fungi which invade the fruit and fructify on the surface in the form of numerous grey or yellowish cushion-like tufts, sometimes irregularly scattered, but often arranged in concentric circles round the point of infection; these tufts, or *sporodochia,* consist of compact branched chains of lemon-shaped, ovoid, or ellipsoid spores (conidia) a type of fungal fructification characteristic of the genus known as *Monilia.**

The change induced in fruits by these particular fungi is not a "rot" in the sense of putrefaction, since the attacked tissues become so permeated with the fungal hyphae that the whole fruit eventually becomes more or less indurated rather than rotten. A fruit so infected, instead of disintegrating, becomes dried up and "mummified". Under normal conditions in the open this desiccation takes place rapidly, not only because moisture is lost from the mechanical rupture of the skin by the fungus fructifications, but also by the activity of the fungus itself in absorbing moisture from the substratum and dispersing it into the air from its own cells by transpiration. Such fruits quickly shrink and dry up to form the "mummies" that are such a conspicuous feature of neglected orchards, particularly in winter when the branches are otherwise naked. On affected apples in storage there may be few or no fungus fructifications, but the browning becomes more intense and may turn eventually to black; the fruit under these conditions loses moisture very slowly and may retain its rounded form for a long time.

Rotting the fruit is not the only damage wrought by the brown rot fungi. The fruit is the only common mode of entry for one species, *Sclerotinia fructigena* (though infection of the flowers by this fungus has been mentioned by some workers, see p. 67), and it may extend from an infected fruit into the spur and branches of the tree. The other two species *S. laxa* and *S. fructicola,* however, infect not only the fruit, but also the flowers, resulting in blossom wilt and eventually cankers; furthermore, the young leaves may become infected, and the fungus extending from them into the shoots causes shoot wilt or wither tip and sometimes cankers on the younger branches.

The sporodochia produce myriads of powdery spores which are dispersed into the air and carried long distances in the wind. Usually the fungus fructifications develop and shed spores within a few days of infection. Thus there may be several generations during the spring and summer.

All the commonly cultivated species of *Pyrus* (apple, pear, quince) and *Prunus* (plum, cherry, peach, nectarine, apricot) are liable to attack by these brown rot fungi. Generally speaking, each of the three fungi is capable of infecting all the host plants mentioned, so that any one of these crops, if infected, may serve as a source of infection for the others. Moreover, certain ornamental species of *Pyrus, Prunus* and related genera are also susceptible to infection by one or more of these fungi, and this fact too must be taken into consideration in relation to control measures.

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* From the Latin *monile,* a necklace.

The effect produced on the fruit by these species of *Monilia* is commonly known as "Brown Rot" in this country, and this term is also now often applied to the other various forms of diseases of fruit trees caused by these fungi. On the Continent the same diseases are referred to as "Rot brun" (France), "Fäulniss der Früchte", "Grind oder Schimmel des Obstes", "Schwarzfäule", "Blüten- und Zweigdurre" (Germany), "Muffa o marciuma dei frutti" (Italy), "Gul frugtskimmel" (Denmark).

Considering the rapid development of the fungi, the ready dispersal of their spores, the varied modes of infection and the wide range of host plants, it is not surprising that the brown rot diseases are widespread and that the yearly losses due to them throughout the temperate regions of the world are enormous.

HISTORY

The earliest record of a brown rot fungus appears to be that of Persoon (1796), who found a fungus producing tufts of moniliform chains of spores on the decaying fruits of *Pyrus* (pear), *Prunus domestica* (plum) and *Amygdalus persica* (peach), and named it *Torula fructigena,* a name used also by Albertini and Schweinitz (1805) and others. In 1801 Persoon changed the name to *Monilia fructigena* and this is still often used for the imperfect or conidial stage of the fungus. Kunze and Schmidt (1817) referred to the fungus as *Oidium fructigenum,* a name used by a number of mycologists in Britain during the nineteenth century, including M. J. Berkeley (1836), Cooke (1871) and W. G. Smith (1885).

Ehrenberg (1818) discovered on apricots a fungus to which he gave the name *Oidium laxum* (later changed to *Monilia laxa* Sacc. & Vogl.). Bonorden (1851) applied the name *Monilia cinerea* to a fungus found on rotting fruit, and this is still commonly used to designate the grey Monilia of plums and cherries.

Woronin (1900) published a detailed description of *Monilia fructigena* and *M. cinerea,* showing that they were distinct species, and for some years it was customary to regard the brown rot fungi of Europe as three species, viz. *Monilia fructigena* (considered by some to be confined, or almost so, to pome fruits, but now known to be prevalent also on stone fruits), *M. cinerea* (on plums and cherries), and *M. laxa* (on apricots).

Schröter (1893), from analogy with other species of *Monilia* known to have a perfect stage, i.e. an ascigerous stage, concluded that *M. fructigena* and *M. cinerea* were each the conidial stage of an ascomycete, and named the fungi *Sclerotinia fructigena* and *S. cinerea,* although the Sclerotinia stage had not then been found. Later, the perfect stages of *Monilia fructigena* and of *M. laxa* were discovered and described by Aderhold and Ruhland (1905), who also gave a description of *Sclerotinia cinerea,* basing this, however, on preserved material from America, assuming it to be the perfect stage of *Monilia cinerea* of Europe, an assumption that more recent work has shown to be inadmissible.

The connection between *M. cinerea* of Europe and an ascomycete was established by Wormald (1921), who pointed out that *Sclerotinia laxa* and *S. cinerea* were indistinguishable in both ascigerous and conidial stages.

The brown rot fungus common in America has also been submitted to similar vicissitudes of nomenclature. For some years it was considered to be identical with *S. fructigena* of Europe, and then with *S. cinerea*. When it was found to be a different species altogether it received the name *S. americana* and more recently that of *S. fructicola* (see p. 22 under "Nomenclature").

That the fruit-rotting *Monilias* were of economic importance was recognized by von Thümen (1875). Soon afterwards serious outbreaks of fruit tree diseases caused by these fungi attracted attention on the Continent, and during the last decade of the nineteenth century numerous articles on the subject were contributed by well-known European mycologists, particularly in Germany, e.g. Behrens (1898), Frank and Krüger (1899), Sorauer (1899), and Müller-Thurgau (1900). During the present century, workers in every European country have studied and described their own particular brown rot problems. Meanwhile, many investigators in other parts of the world, especially in North America, Australia and New Zealand have carried on the work on these diseases.

Continent of Europe

As indicated above, the pioneer work (to the end of the nineteenth century) on the brown rot diseases in Europe was mostly that of mycologists in Germany, and of Woronin whose classical work appeared in 1900. Observations on the Continent since 1900 have been continued with particular reference to blossom wilt and of the resulting killing-back of twigs of Morello cherries in Germany, and of apricot in the Rhône valley and around the Black Sea. Important contributions to our knowledge of brown rot have been published in Germany, (Aderhold & Ruhland, 1905 ; and more recently Mittmann-Maier, 1940*a*, 1940*b* and Maier, 1942*a*, 1942*b*): France, (Killian, 1921 ; Joëssel, 1932*a*, 1932*b* ; Chabrolin, 1924): Switzerland (Faes & Staehelin, 1925*a*, 1925*b*): Sweden (Eriksson, 1913): Italy (Manaresi, 1920 ; Peglion, 1921 ; Pesante, 1938): Russia (Voronichin, 1914 ; Balakhonoff, 1931*b* ; Solkina, 1931 ; Youganova, 1946): Holland (Ritzema Bos, 1904 ; Westerdijk, 1912).

Only a few of the names associated with brown rot investigations are mentioned above ; others will be given later in the section on Geographical Distribution. In other European countries, too, the diseases have been recorded and control measures recommended.

The British Isles

In 1836, the Rev. M. J. Berkeley recorded *Oidium fructigenum* on decayed pears, apples, plums, etc., exposed to wet, and in 1857 he wrote with regard to the same fungus that it formed concentric patches on apples, pears, plums, etc., producing very rapid decay. Again, in 1864, he described a destructive disease of apricots caused by a little greyish mould, *Oidium fructigenum*. Cooke (1871) mentioned that the same fungus was found on decayed pears, apples, plums, etc. In 1885, W. G. Smith remarked that the fungus was often extremely destructive to stored apples when kept in damp or unventilated store-rooms and the same pest destroyed pears, plums and fruits. He probably had before him what is now known as Black Apple disease, for, he says that the fungus had, in some instances, the power of changing the skin of the apple to jet black. It would appear that his

"*Oidium fructigenum*" consisted really of the two fungi *Monilia fructigena* and *Monilia cinerea*, for, he states that the warts were generally grey in colour, varying, however, in tint from cream to fawn, or different shades of grey. Massee (1893) refers to the fungus causing brown rot as *Monilia fructigena* and later (1895) following Schröter, as *Sclerotinia fructigena*.

Early in the present century the brown rot diseases began to receive more notice. Brief descriptions of these diseases were given by Massee (1902, 1903) and by Cooke (1906). Massee & Crossland (1905) mentioned that *S. fructigena* was a very destructive parasite on the fruit of apple, pear, apricot, plum, cherry, etc.

Blossom infection in Morello cherries was described by Salmon (1907), who noted that those branches whose flowers have been 'struck' die, the fungus gradually killing the wood. From field experiments he found that diseased trees can be quickly restored to health by spraying with Bordeaux mixture. Later (1910*a*, 1910*b*, 1913) he also drew attention to severe damage to apple trees resulting from Brown Rot Canker.

As the losses from brown rot diseases were by that time assuming serious proportions, many popular articles appeared in the Press warning growers of the dangers incurred in neglecting control measures. Up to that time these diseases in Britain were all attributed to one fungus *Monilia* (*Sclerotinia*) *fructigena*. In 1915, Cayley published a short description of the brown rot diseases, based chiefly on work carried out abroad, and mentioned three fungi, viz., *Sclerotinia fructigena*, as mostly confined to pome fruit trees—*S. cinerea* on plum and cherries, and *S. laxa* on apricots.

Heavy losses from blossom wilt and brown rot canker in apple trees round about 1915 stimulated a more detailed investigation, and Wormald (1917–1933) published a series of papers on this and other brown rot diseases, calling attention to the distinguishing characters of *S. fructigena*, *S. cinerea* and the American brown rot fungus, in respect of their morphology, parasitism and geographical distribution. *S. cinerea** (not *S. fructigena*) was shown to be the cause of blossom wilts, and it was found that blossom wilt of apples was induced by a specialized form that was referred to as *S. cinerea* forma *mali*, a conclusion confirmed later by Boyle, Murphy and Cummins (1928).

Various aspects of these diseases and the fungi concerned have been studied by other workers in Britain in recent years, viz., Black Apple disease (Spinks, 1916, 1917 ; Drummond, 1934 ; Wormald, 1945) : a Core Rot of apples (Dowson, 1925, 1926): field trials (Lees & Briton-Jones, 1925 ; R. W. Marsh, 1947 ; Byrde, 1949, 1950): indirect control of brown rot by routine spraying against apple Scab (Grubb, 1921 ; M. H. Moore, 1932): the fungicidal action of sulphur on conidia of *Monilia fructigena* (Barker, Gimingham & Wiltshire, 1920 ; R. W. Marsh, 1929): brown rot on imported fruit (Kidd & Tomkins, 1928): cultural and physiological studies (Wiltshire, 1920 ; Hall, 1933 ; Vasudeva, 1930*a*, 1930*b* ; Horne, 1933 ; Menon, 1934 ; Dickson, 1939): leaf infection (Drummond, 1934): and the occurrence of *Monilia fructigena* on cultivated hazel nuts (Wormald, 1944 ; M. H. Moore, 1950*a*).

In Eire, Boyle, Murphy and Cummins (1928) made detailed observations on blossom wilt of apple trees and wither tip of plum trees.

America

The early history of brown rot in the United States is well described by Roberts and Dunegan (1932). Peck (1881) is usually quoted as giving the first adequate description of the disease, but Roberts and Dunegan present evidence to show that brown rot must have been troublesome in the first half of the nineteenth century, and they quote Kirtland who published an excellent description of plum brown rot in 1885 and stated he had known the disease for thirty years. After the publication of Peck's account, papers on brown rot appeared in increasing numbers, including, in the 1880's those by Arthur (1886), E. F. Smith (1889), and Galloway (1889).

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* Referred to in this bulletin as *S. laxa*.

Numerous papers have been devoted to various aspects of brown rot—control measures, nomenclature of the fungi concerned, their physiology and their mode of infection, losses from brown rot on the trees and during storage and marketing. Laboratory methods of study have been chiefly concerned with spore germination tests, as described in the series of papers published from the Boyce Thompson Institute, but attention has also been given to the effect of antibiotics using *Sclerotinia fructicola* as one of the test fungi.

In Canada work in the rich fruit-growing region of the Niagara Peninsula has been chiefly concerned with control measures in peach orchards using sulphur preparations—see G. H. Berkeley (1927, 1928, 1929)—and Willison (1931–1944), and Eastham (1925, 1929, 1935*a*, 1935*b*), have studied the disease in British Columbia.

In South America brown rot (*S. fructicola*) has been reported from Argentina (Soriano, 1928 ; Fresa, 1945), Brazil (Gonçalves, 1939) and Peru (Bazán de Segura, 1946).

Australia and New Zealand

Important papers have appeared in Australia and New Zealand, mostly with reference to control measures in stone fruit trees. Harrison (1922–1935*b*) wrote a series of articles on the nomenclature of the brown rot fungus in Australia, and in New Zealand Cunningham (1922–1934) made biological observations and Curtis (1928, 1929) studied the morphology of the stone fruits in relation to infection.

Other Regions

During the present century observations have been made on brown rot in African and Asiatic countries and the presence of the fungi in those regions has been recorded, while original articles have been published in Japan (Takahashi, 1911 ; Miura, 1929).

GEOGRAPHICAL DISTRIBUTION OF THE BROWN ROT FUNGI

The fungi causing the brown rot diseases of rosaceous plants are not uniformly distributed throughout those regions where fruit trees are cultivated. Comparisons made by a number of plant pathologists of specimens examined, of cultures exchanged from various regions, and a study of the field records from many fruit-growing areas show that their distribution is as shown below:

EUROPE

Austria. Jaap (1908) mentions *Monilia fructigena* on *Pyrus spinosa*. Köck (1910) records observations on varietal susceptibility in 27 varieties of cherry, the Grosse Lange Lotkirsche being very susceptible. Brož (1915) says that fruit rot had been known for a long time, but that blossom wilt and the attendant dying-back of branches had only recently attracted much attention ; he refers to its occurrence on apple, assuming that the causal organism was *Sclerotinia fructigena.* Wahl (1925, 1926) states that cultivation of stone-fruit trees is seriously threatened by the steady increase in the prevalence of Sclerotinia rot, particularly in apricot orchards. More recently Schmidt (1949) finds that infection by *S. fructigena* and *S. cinerea* is serious and Wenzl (1950*b*), describing apricot necrosis, includes a tip necrosis due to blossom or fruit infection by *S. laxa.*

Bavaria. The brown rot diseases appear to have received little attention here. Sprau (1948), however, refers to *Monilia fructigena* on apples in storage.

Belgium. In Belgium the brown rot diseases have not been investigated in any great detail. *Sclerotinia fructigena* and *S. cinerea* are included, in a list of new species, by Marchall and Marchall (1921) for the region round Gembloux. More recently they have been noted by Hallemans (1946), and about the same time Estienne and Soenen (1946) remarked that acid cherry plantations were being destroyed by brown rot infection, and that the yield of sweet cherries was reduced by 30 per cent.

Bulgaria. Malkoff (1908) recorded *Monilia fructigena* on apple, pear and plum, and *M. cinerea* on acid cherry. More recently Christoff (1934*a*, 1938) found *M. fructigena* on five species of *Prunus* and on *Cydonia japonica,* and *Monilia cinerea* on *Prunus domestica* and *P. insititia.*

Czechoslovakia. Vielwerth (1938) states that stone fruits are severely attacked by *Sclerotinia fructigena* and *S. laxa,* particularly apricots, so it would appear that both fungi are present in Czechoslovakia.

Denmark. From various notices by Weber and her collaborators, from 1926 to 1938, it is clear that the brown rot fungi cause serious losses in Denmark. *S. fructigena* attacks apples, pears (60 per cent of the ripening pears may be spoiled at times), and hazel nuts, while *S. laxa* is common on cherries, particularly the acid varieties, and plums, and is also recorded as attacking flowers and twigs of *Prunus pseudocerasus* and *P. triloba,* and causing blossom wilt of pears. *Sclerotinia laxa* f. *mali* produces blossom wilt and cankers on apple trees.

Eire. In Eire brown rot of apples and blossom wilt of apple and plum trees are mentioned by Murphy (1926), and blossom wilt of apple and plum and wither tip of plum were investigated and described in detail by Boyle, Murphy and Cummins (1928).

Estonia. Kivilaan (1936) reported that in 1935 *S. fructigena* caused serious infection of certain varieties of apple, and that *S. cinerea* occurred on plum and cherry trees.

Finland. As Woronin (1897, 1900) obtained some of the material for his study of *S. fructigena* and *S. cinerea* from orchards at Viborg, brown rot was probably prevalent there. Blossom wilt of cherries had not attracted notice before 1897, but in that and the following year, trees were seriously damaged.

France. De Jubainville *et al.* (1878) do not mention brown rot at all, so that it would appear that these diseases had not attracted particular attention up to that time. Molliard (1901) writes that although *Monilia fructigena* very frequently attacks pome and stone fruits causing them to become mummified, severe losses like those recorded in America and Germany at that time had not occurred in France. He then describes severe infection of apricot blossoms near Paris, where in an orchard and a neighbouring garden the flowering shoots were destroyed, the crop failing completely.

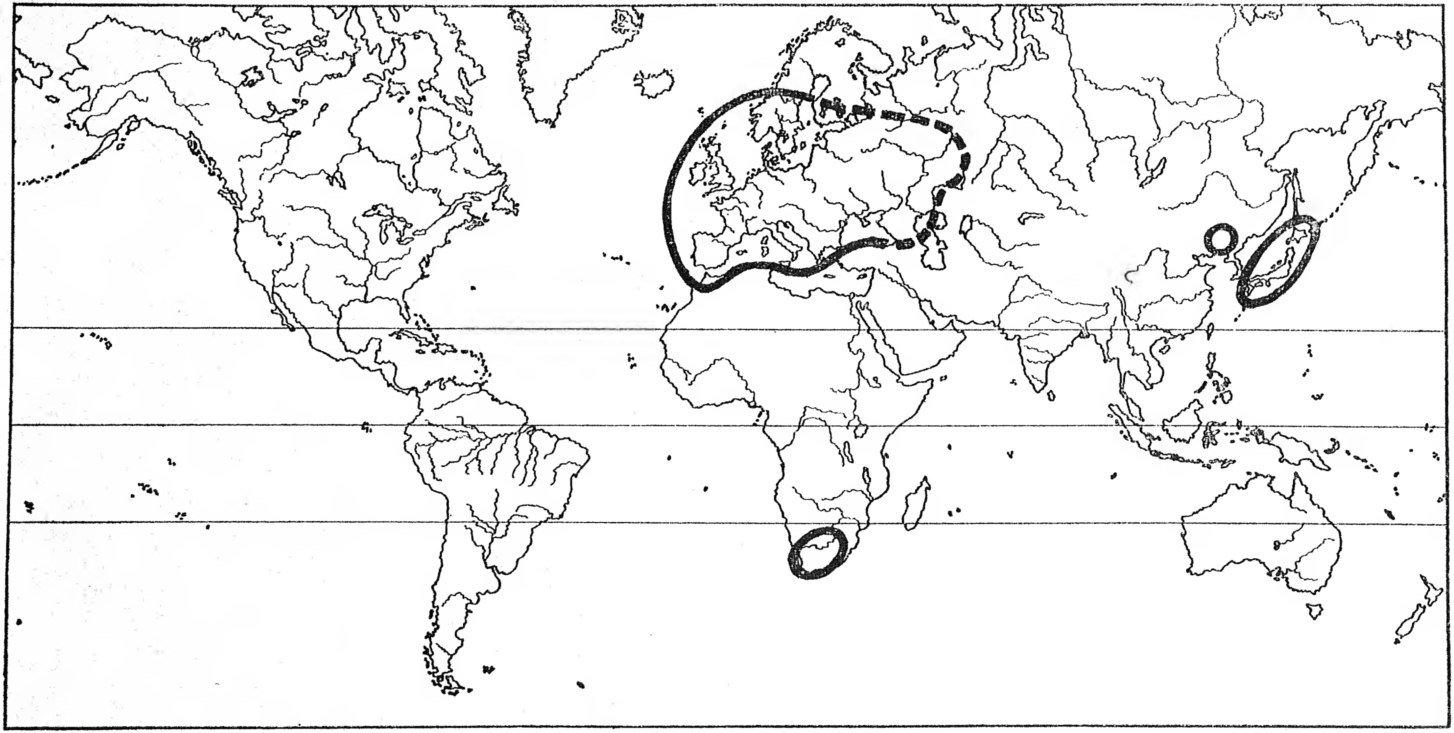

FIG. 1. Distribution of *Sclerotinia fructigena*

Hosts: Apple, pear, plum, cherry, etc.

(Distribution Map No. 22 of the Commonwealth Mycological Institute, Revised to 2.xi.1942)

Delacroix and Maublanc (1909) refer to brown rot of fruit as of common occurrence and mention *Stromatinia fructigena, S. cinerea* (mostly on stone fruits), and *S. laxa* (on apricot). They say, however, that the infection of flowers " est peu repandue en France ". About 1920 blossom wilt, accompanied by the dying-back of the flowering branches of apricots, particularly in the Rhône valley, was causing considerable losses as testified by Chabrolin (1922, 1923*a*, 1923*b*, 1924) and by Joëssel (1928–32). Recently Chaboussou (1945) described *Sclerotinia fructigena* as being particularly destructive on d'Ente prune and on nectarines.

Germany. It is evident, from the published accounts to which reference has already been made, that brown rot of pome and stone fruits, and blossom wilt of stone fruit trees are prevalent in Germany. Brown rot of apples and pears is caused by *Sclerotinia fructigena,* as in England, and blossom wilt of stone fruit trees by *S. laxa.* No serious blossom wilt attacks on apple have been described so it would appear that *S. laxa* f. *mali,* if it does occur in Germany, does not cause the serious infections reported in England.

Greece. In Greece *Monilia cinerea* is associated with blossom infection and gummosis of drupaceous fruit trees, and the dying-back of the shoots of almonds following blossom wilt causes serious losses as recorded by Anagnostopoulos (1930, 1933). Sarejanni (1935, 1936, 1939) states that the disease reduces the almond crop in Crete by nine-tenths ; he mentions *Sclerotinia fructigena* on apples and pears and *S. cinerea* on loquat.

Holland. The brown rot diseases of fruit in Holland, particularly the killing of twigs of Morello cherry and peach trees, were described by Ritzema Bos (1896, 1898, 1904) who attributed infection to *Monilia fructigena,* and did not recognize *M. cinerea* as distinct. Schwarz (1922) describes twig die-back of peaches, *M. cinerea* being one of the fungi concerned, and Berkhout (1923) records various observations on *M. fructigena* and *M. cinerea.* Brown rot of pome and stone fruits (apple, pear, cherry, Morello cherry, plum, apricot and peach) is mentioned by Goossens (1938). An apothecial form found on cherries was described by Westerdijk (1912).

Hungary. *Sclerotinia laxa* appears to be widespread and destructive on apricots and acid cherries in Hungary, as reported by Istvánffi (1906), by Schilberszky (1933), and by Husz (1936) who stated that Monilia blossom blight is a limiting factor in growing apricots, and sometimes took the character of a destructive epiphytotic.

Italy. Comes (1891) mentions *S. fructigena* as causing fruit rot, but makes no mention of blossom wilt in Italy, so it would appear to have been of little importance there at that time.

Montemartini (1924, 1936) mentions *S. fructigena* (for Sicily) as not being controlled by any of the treatments tried, and refers to infection occurring through injuries caused by *Rhynchites bacchus.* More recently, however, Cristinzio (1934) describes blossom wilt of various kinds of fruit trees in southern Italy, and Bevilacqua (1935) severe blossom wilt on cherries in the coastal regions of Istria so that *Sclerotinia laxa* would appear to be well established in certain parts of Italy by that time. Experimental work on the brown rot fungi has been carried out by Peglion (1921), and Pesante (1935).

Latvia. Smarods (1930) records *S. fructigena* on apple and pear, and *S. cinerea* on plum and cherry.

Norway. Conditions in Norway appear to favour brown rot infection. Reuter (1898) mentions *Monilia fructigena* on plums in a garden, but details

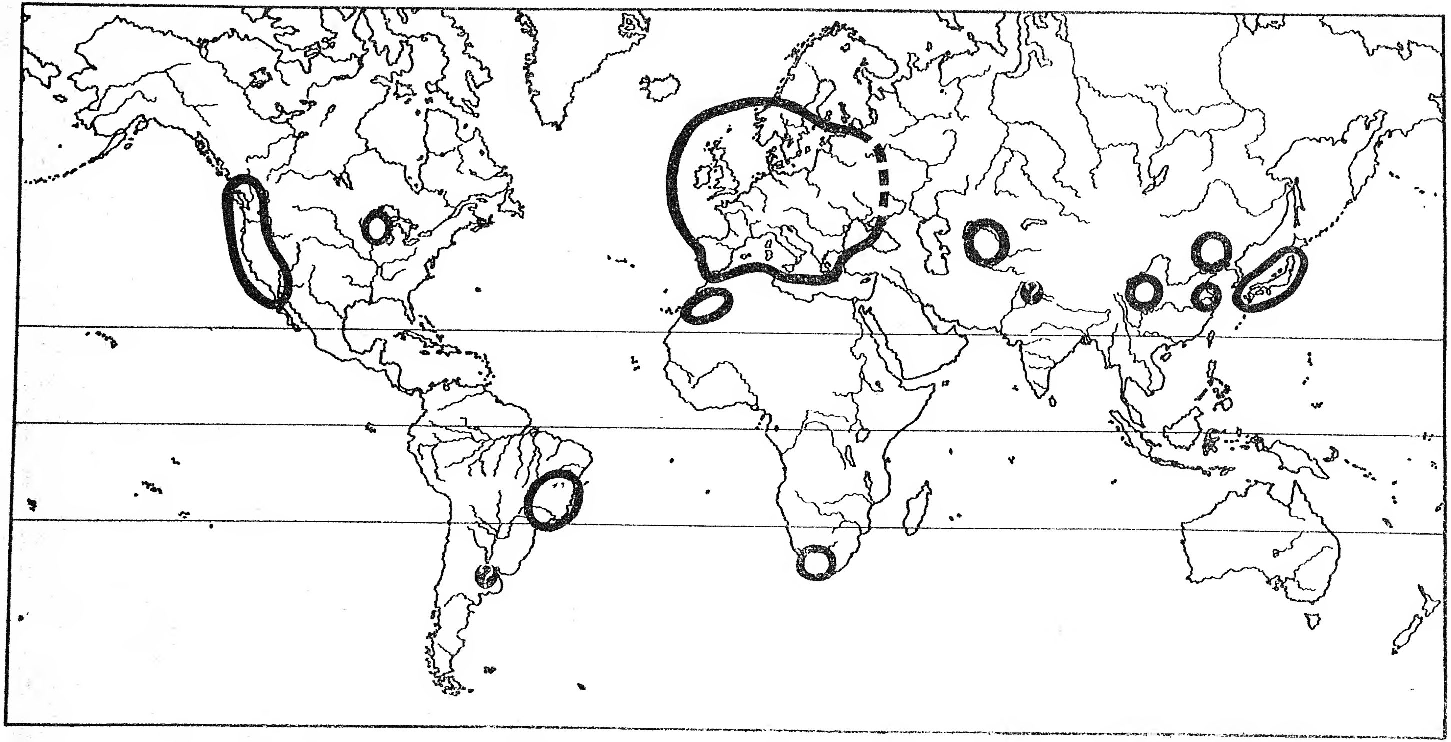

FIG. 2. Distribution of *Sclerotinia laxa* Aderh. & Ruhl.

Hosts: Plum, cherry, apricot, pear, etc.

(Distribution Map No. 44 of the Commonwealth Mycological Institute, 2nd Edition, Revised to 1.v.1948)

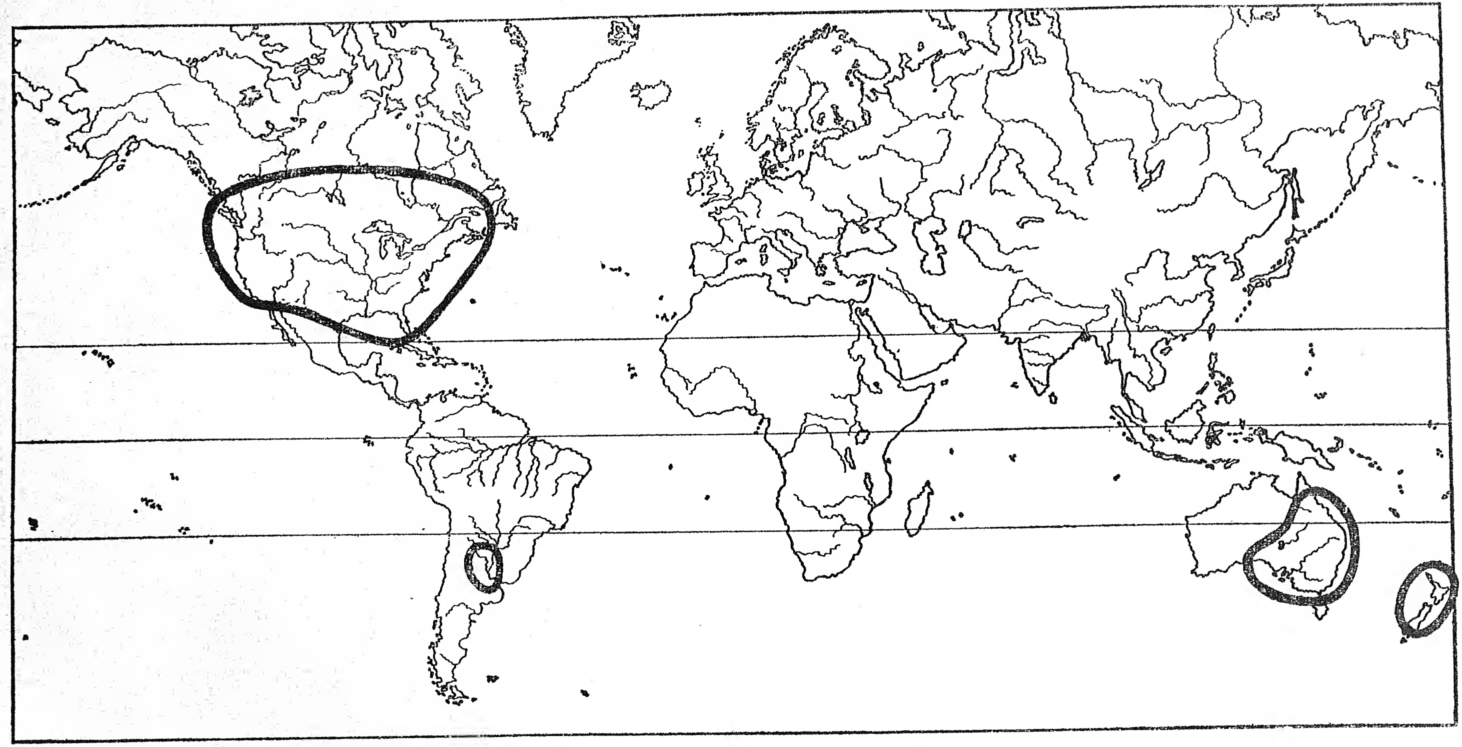

FIG. 3. Distribution of *Sclerotinia fructicola* (Wint.) Rehm

Hosts: Plum, peach, apricot, cherry, etc.

(Distribution Map No. 50 of the Commonwealth Mycological Institute, 2nd Edition, Revised to 1. viii.1947)

of more widespread attacks are given by Jørstad (1923, 1938, 1945) who noted varietal differences in susceptibility. He reports *Sclerotinia fructigena* on apples, pears, plums, and sweet cherries, and *S. laxa* on apple, pear, sweet and acid cherries, plum and *Prunus triloba, Sclerotinia laxa* being found in the east of the country on the Cellini apple.

Poland. Garbowski and Juraszkówna (1933) report *Monilia cinerea* on plum, sweet and acid cherries, *Prunus* sp. and *P. triloba; Monilia fructigena* on apple, pear, plum and *Pyrus mala baccata: Monilia laxa* on apricot: and *M. linhartiana* on *Prunus padus.*

Portugal. Little or no work on the brown rot fungi appears to have been carried out in Portugal, but both *Sclerotinia fructigena* and *S. laxa* occur in that country, as is shown by the fact that on fruit imported into the United States from Portugal, *S. fructigena* was found on an apple in 1945, and *S. laxa* on cherries in 1943.

Rumania. In a preliminary account of observations during many years in Bessarabia and Bukovina on fruit tree diseases Veresciaghin (1940) records *S. fructigena* on apples, *S. cinerea* on plums and *S. laxa* on apricots. Savulescu *et al.* (1941) report finding *S. fructigena* on apple, *S. cinerea* on cherry and *S. laxa* on apricot.

Spain. *Sclerotinia fructigena* and *S. laxa* are both found in Spain and are mentioned by Sardiña (1928): by Unamuno (1929) who noted *M. fructigena* on plums (*Prunus domestica*) as a new host for Spain: Benlloch (1931) who reports *Sclerotinia fructigena* on apples and pears, *S. cinerea* on plums, cherries, and peaches, and *S. laxa* on apricots: and by Gallástegui (1942) who finds cider apples infected with *Monilia fructigena* which is said to attack leaf buds, blossoms and fruit.

Sweden. Wulff (1906) describes *Monilia cinerea* particularly as causing blossom wilt of acid cherry and fruit rot of plum. An important article on brown rot by Eriksson (1913) is an account, with illustrations, of blossom wilt of apple so that it would seem that the cause was *Sclerotinia laxa* f. *mali* although Eriksson includes all forms under *Monilia fructigena.* Henning (1922) found brown rot very prevalent on the apple variety Signe Tillisch, and Lindfors and Holmberg (1941) mention brown rot of apple, pear, plum, cherry and apricot, with *Sclerotinia laxa* on *Prunus triloba.*

Switzerland. Müller-Thurgau and Osterwalder (1924) mention, as occurring in Switzerland, *Sclerotinia fructigena* on apple, pear, and quince, *S. cinerea* on cherries and damsons, and *S. laxa* on apricots. Soon afterwards a series of articles by Faes and Staehelin (1924, 1925*a,* 1927) describe the fungi under the same three names with special reference to the severe infection of apricots in Valais, and the importance of this disease is emphasized by Faes and Tonduz (1923, 1925, 1926).

United Kingdom. In England, *Sclerotinia fructigena* is common not only on apples, pears and quinces, but also on stone fruits, causing fruit rot, and, on apple trees, sometimes producing cankers. *S. laxa* is general as a fruit rot fungus of stone fruits (rarely of apples and pears), but is more serious as the cause of blossom wilt of plums, cherries and pears. *S. laxa* f. *mali* is the cause of blossom wilt of apple trees; it is common in the southern counties and it occurs in Scotland.

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In Scotland, the records of Alcock and Foister (1931) and of Dennis and Foister (1942) indicate that *S. fructigena* infects the fruit of apple, pear and peach, while *S. laxa* infects the fruit of plum, cherry and peach; causes blossom wilt of pear, plum and cherry, and wither tip of plum, of *Prunus padus* and of *P. besseyi,* and *Sclerotinia laxa* f. *mali* causes blossom wilt of apple.

U.S.S.R. As Woronin (1900) obtained some of his material from Kiev for his much-quoted publication on the brown rot fungi it may be assumed that both *S. fructigena* and *S. cinerea* were prevalent at that time in the Ukraine. Since that time severe losses from brown rot infection have been reported in southern Russia, particularly in relation to their association with the moist air and the sea mists in spring in the coastal regions of the Black Sea. Losses of up to 36 per cent in plums and 38 per cent in apples were reported by Voronichin (1914), and a few years later Truesdell (1922), who visited the Crimea, wrote of neglected orchards where *S. fructigena* and *S. cinerea* were both in evidence. Andreyeff (1924) also mentions neglected orchards where apples and pears suffered severely from these two fungi, and Gerassimenko (1926) writes of *Monilia cinerea* causing fruit rot of cherries in the Ukraine.

Bondartzev (1931), too, mentions the brown rot fungi and also refers to the great damage caused in apricot orchards in the Crimea by *Sclerotinia laxa,* sometimes whole trees being affected. These observations on apricot brown rot are supplemented by Strelin (1926) and particularly by Balakhonoff (1930, 1931*b*) who gives a detailed account of the disease and says it was so severe that the cultivation of cherries in places, and of apricots almost everywhere, was dying out.

Other observers of the brown rot diseases in Russia are Lopatin (1936) who describes an apple blossom wilt, attributing it to *S. fructigena,* but this is probably an error for from his description it is more probably *S. laxa* f. *mali,* and Ovtshinnikova (1937) who mentions *Monilia fructigena* and *M. cinerea* as occurring in the Krasnodar region of the North Caucasus. Nagorny and Issarlischwili (1929) record *M. fructigena* on grapes in Georgia.

Reports issued from the Sukhum Experimental Station, Tiflis, Georgia, in 1914 and 1915 mention *M. fructigena* on apple, pear, plum, peach and Japanese plum, and *M. cinerea* on flowers and fruit of plums and sweet and acid cherries.

The apricot disease is again mentioned by Youganova (1946) who refers to the severe infection of buds, flowers, twigs and fruits of apricots.

Yugoslavia. According to Škorič, *Sclerotinia fructigena* occurs every year on apples and pears and in some years considerable damage is caused to cherry trees by *S. cinerea.* Mijuskhovits (1950) reports *Monilia fructigena* on apple, pear and plum and *M. cinerea* on cherry, peach, and apricot in Montenegro, and Perishits (1950) *Sclerotinia fructigena* on apple and pear and *S. cinerea* on plum.

ASIA

China. Wei (1941) in notes on diseases of stone fruits found in the Chengtu market during 1936 to 1940 records *Sclerotinia laxa* on apricot and peach. Ling (1948) in his host index of the parasitic fungi of Szechwan includes *S. laxa* on *Prunus armeniaca, P. persica* and *Eriobotrya japonica.*

India, Pakistan and Kashmir. From information received from a member of the staff of the Agricultural Department, Srinagar, Ferraris (1928*a*) records that brown rot (*Monilia cinerea*) occurs on peaches in Kashmir. Butler and Bisby (1931) refer to this paper but doubt the correctness of this determination.

According to notes by Sattar (1940) and Hafiz (1946) brown rot (*Sclerotinia fructigena*) of apples and pears is common in the Punjab and causes heavy losses ; Varma (1945) writes that the Japanese plum in the hill orchards of Patiala State is susceptible to brown rot.

Iran. Esfandiari (1947, 1949) reports *Monilia fructigena* on pears along the shores of the Caspian Sea, in one place causing 30 per cent loss ; it is said to occur on quinces also. Pome and stone fruit trees, however, are not abundant in this region, because of its subtropical climate, and they are grown only in small gardens.

Japan and Manchuria. Takahashi (1911) in a *résumé* of an original Japanese paper on Sclerotinia diseases of rosaceous fruit trees in Japan writes that *Sclerotinia cinerea* occurs in Hokkaido on the blossoms of apricot, *Prunus mume,* and *P. tomentosa,* and on the fruits of cherry and apricot and that *Sclerotinia fructigena* is quite common in Japan on the fruits of apple and pear and also appears on the ripe fruits of quince, cherry, apricot, peach, plum, *Prunus mume, P. pseudo-cerasus,* grape, and *Elaeagnus macrophylla.*

Wormald (1927*a*) examined material sent to him by Michuya Miura and confirmed the presence of *Sclerotinia fructigena* on peach from Japan, and pear from S. Manchuria, and *S. laxa* on cherry and Chinese plum from Manchuria.

Miura (1929) mentions *S. fructigena* as occurring in Japan and Manchuria, and *S. laxa* on unripe fruit of *Prunus manshurica* (the Manchurian apricot) on ripe cherries and peaches and unripe apricots, in Manchuria, and on apples in Japan and Manchuria.

Other Sclerotinias described as infecting rosaceous hosts are mentioned later (see p. 22).

Syria. According to Najjar (1947) brown rot of apricots [? *Sclerotinia laxa*] is a serious disease in Syria, causing twig blight and branch cankers ; it rarely infects apricot fruits but is prevalent on peaches.

Turkey. Bremer *et al* (1947) record *S. fructigena* on apple, pear, quince and peach, and *S. laxa* on apricots, while Karel (1949) states that brown rot causes great damage to all *Prunus* spp., especially apricot.

U.S.S.R. (Asian). Zaprometoff (1925) recorded *Sclerotinia cinerea* on sweet cherry in the Tashkent region of Turkestan ; he stated that brown rot of apples, *S. fructigena,* was not found in that region.

AFRICA

Morocco. Both *S. fructigena* and *S. laxa* occur in French Morocco. Bouhelier (1936) recorded *S. fructigena* on apple and pear, and *S. cinerea* on almond, peach, plum and apricot. Malençon and Delécluse (1937), in a list of pathogenic fungi, include *S. laxa* on apricot. More recently workers at the Bureau de la Défense des Végétaux (1941), mention *Monilia cinerea* as occurring particularly on peach and almond infecting flowers, leaves and fruit, though they say " Les feuilles sont rarement infecties d'une façon direct ".

South Africa. Brown rot of rosaceous fruits is apparently seldom found in the fruit-growing regions of South Africa, doubtless as a result of the dry climate. Doidge (1919) mentioned *Sclerotinia fructigena* as causing " fruit mould " (but with no evidence as to the identity of the fungus) and wrote that happily South African conditions do not seem favourable to the fungus causing the disease, and it is only in exceptionally wet seasons that it has been noticed as causing any appreciable damage.

Doidge and Bottomley (1931) in a revised list of plant diseases occurring in South Africa, include *S. fructigena* on apple (occasional occurrence on twigs), apricot (on mummified and fresh fruit; only reported during one exceptionally wet season in the Western Cape Province), peach (rare), and plum (uncommon), and *S. cinerea* on cherry (in the Cape Province only). In a more recent list of South African fungi and lichens Doidge (1950) includes *S. fructicola* (?Monilia form only) on *Prunus persica* (fruit) and on *P. salicina* and hybrids; if the fungus is indeed *Sclerotinia fructicola* it would seem that it has been introduced into South Africa from either America or Australasia.

NORTH AMERICA

Canada. The distribution of the brown rot fungi in Canada is, in general, similar to that in the United States (see below). *Sclerotinia fructicola* is very destructive in the east, particularly on peaches in the Niagara Peninsula. In Vancouver Island another fungus reported by Eastham (1925, 1935*a*), at first referred to as *Monilia oregonensis*, proved to be *Sclerotinia laxa* as found in the Pacific coastal regions of the United States.

Crowell and Lavalee (1942) give the detailed distribution of *Monilinia fructicola* (Wint.) Honey on *Malus pumila, Prunus armeniaca, P. avium. P. cerasus, P. besseyi, P. domestica, P. nigra* and *P. persica.*

United States. The brown rot fungus distributed throughout the fruit-growing areas of the United States is *Sclerotinia fructicola.* In the Pacific coastal regions another brown rot fungus was found which at first was thought to be a new species for it was different in some respects from *S. fructicola* and was named *Monilia oregonensis.* Ezekial's observations (1924*b*) suggested, however, that it was identical with *S. laxa* f. *pruni* of Europe, and this was confirmed by Wormald (1927), Hewitt and Leach (1939) and Huber and Baur (1941). Although most frequent in the western states—Washington, Oregon, California—where it was first noted, it has more recently been recorded in Wisconsin by Keitt *et al* (1943). It is probable that attacks by this fungus (referred to as *Sclerotinia cinerea*) were being described by Howard (1922) in California when he wrote of the worst disease of the coastal region which seemed to be on the increase not only along the coast but in rather remote coastal valleys and for almost a hundred miles up the Sacramento River, and attacked both fruit and flowers.

SOUTH AMERICA

The brown rot fungus of South America appears to be *Sclerotinia fructicola* when the identification is based on cultural tests. Where it is referred to as *S. cinerea,* tests for determination are not mentioned.

Argentina. Soriano (1928) says that *S. cinerea* causes heavy damage to peaches. Later, Fresa (1945) refers to fruit rot of peaches and plum and occasional blossom wilt of peach as caused by *S. fructicola,* identified by cultural tests; he mentions losses as high as 60 per cent in years of severe infection.

Brazil. Gonçalves (1939) writes that *S. cinerea* is widespread and destructive to peaches in Brazil.

Guatemala. Muller (1950) records *S. cinerea* as causing loss in fruit.

Peru. A fungus isolated from diseased peaches from the Department of Huánoco, Peru, was identified by Bazán de Segura (1946) as *S. fructicola* from its morphology when grown on potato dextrose agar and by the effects induced on the host plant.

AUSTRALASIA

Australia. The history of the cultivation of rosaceous fruits in Australia and of brown rot as causing serious reductions in yields is admirably described by Harrison (1928). He gives a map showing the geographical range of brown rot in Australia in 1928 as extending, in a coastal strip averaging about 400 miles wide, from the Stanhope area in the south-east corner of Queensland into Victoria, with two small areas in Tasmania. Until comparatively recently the fungus was referred to as *Monilia fructigena.* Thus McAlpine (1902) uses that name in describing the damage to apricots, peaches, and cherries in Victoria and South Australia, though it seems doubtful whether it really occurred in South Australia (see Harrison, *loc. cit.* p. 106); apparently it is not found in West Australia.

Harrison, from comparison with strains obtained from America and Europe, concludes that the brown rot fungus of Australia is *Sclerotinia fructicola.* He isolated the organism in New South Wales from the fruits of apple, apricot, blackberry, cherry, nectarine, peach (commercial and ornamental), pear, plum (English, Japanese and prune) and quince, and artificially infected fruits of grape, loquat, persimmon and tomato. It was also isolated from twigs of apricot, nectarine, peach, plum and quince, and from blossoms of apricot, nectarine, peach and plum, and cankers were noted on limbs of apricot nectarine and peach trees. Other aspects of the brown rot diseases are discussed by the same author, e.g. taxonomy and nomenclature (1933) and the apothecia of *S. fructicola* in Australia (1922, 1935*a*, 1935*b*).

In a list of plant diseases recorded in New South Wales, Noble *et al.* (1934) mention *S. fructicola* as found on apple in 1910 [pear is not included], on peach prior to 1904, on apricot in 1916, on cherry in 1893, on plum in 1910; on the stone fruits it is indicated as a disease of major importance. In a supplement, issued 1937, they add its occurrence on a flowering peach. In 1937 Noble *et al.* say that brown rot continues to be the principal source of loss in stone fruit crops.

Simmonds (1938) writes that, in Queensland, plum and nectarines suffer most from brown rot (*S. fructicola*), which also occurs on apricots, cherries, and occasionally pome fruits.

Other workers in Australia have been mostly concerned with observations (particularly with reference to the losses caused) and control measures, e.g. Ward (1926); Fish (1927); Hutton (1947*a*, 1947*b*); Wade (1949).

New Zealand. The conidial stage of a brown rot fungus, referred to at the the time as *Sclerotinia fructigena* was first recorded for New Zealand by Kirk (1905). The ascigerous stage was found by Cunningham (1922*b*, 1922*c*, 1922*d*) who also gives (1925*a*, 1925*b*) a detailed description of the disease as found in New Zealand. He calls the fungus *S. cinerea* Schroeter, but uses *S. americana* Norton & Ezekiel as a synonym without distinguishing between the two. Cunningham states emphatically that *S. fructigena* does not occur in New Zealand.

Wormald (1927) who received material from New Zealand in 1921 isolated *S. americana* [*S. fructicola*] from apricot, peach and plum mummies and peach twigs. There is no evidence that either *S. fructigena* or *S. laxa* is present in New Zealand.

It seems clear then that up to the present time the only brown rot fungus present in Australasia is *Sclerotinia fructicola.*

GENERAL DISTRIBUTION

The chief regions of distribution of the brown rot fungi as indicated by the preceding review (see also Figs. 1, 2 and 3 ; pp. 7-10) are :

S. fructigena.	Europe, Japan, Manchuria, Turkey, Uzbekistan.
S. laxa.	Europe, Japan, Manchuria, Turkey, Uzbekistan and the Pacific coastal regions of N. America.
S. laxa forma *mali.*	Europe.
S. fructicola.	N. America, S. America, Australia and New Zealand.

The most striking features of this distribution are the absence of *S. fructigena* from America and Australasia, and of *S. fructicola* from the Old World. *S. laxa* has not yet been recorded from Australia or New Zealand, and this also is a fact to be noted. The following points should therefore be carefully considered with reference to attempts to prevent any one of these fungi from becoming established in a region where it has not yet been notified.

Large quantities of apples are exported from North America, Australia and New Zealand, regions where brown rot of apples is not serious, probably because *S. fructigena* is absent. From an economic standpoint it is important that this fungus should not become established there.

Sclerotinia laxa also is absent from those countries except in the western parts of N. America. It is another undesirable parasite, particularly because it infects the flowers and causes blossom wilt. *S. laxa* f. *mali* too might be troublesome if introduced into the apple orchards of America and Australasia.

With regard to the British Isles and the rest of Europe, the chief feature of interest is the absence of *S. fructicola,* a fungus very destructive to stone-fruits. Whether its introduction into Europe would be a serious matter cannot be predicted with certainty, but it is clearly undesirable that it should become established. It has been found on more than one occasion on fruit imported into this country from America. Evidence indicates that it produces conidia more freely than *S. laxa,* and its ascigerous form, which disperses innumerable ascospores at the time the trees are coming into blossom, is a serious menace to the cultivation of stone fruits.

It must be borne in mind that there is a real risk of introducing a species into a country where it is not at present found. That this is so is shown by the fact that infected imported specimens have been intercepted on several occasions.

In the U.S. Department of Agriculture's lists of intercepted plant pests (1944, 1946, 1950) the following items have appeared:

Monilinia cinerea on cherries from Portugal in 1943.
Sclerotinia fructigena on apple in stores from Portugal in 1945.
Monilia fructigena on apple from Sweden in 1948.

In trials in which peaches were dusted with sulphur against *Sclerotinia fructicola* in Canada and sent as a consignment to East Malling to be examined by the present writer for degree of infection on arrival, some of the fruit showed brown rot when the cases were opened. Kidd and Tomkins (1928) found *Sclerotinia americana* (syn. *S. fructicola*) in peaches imported from Georgia and *Monilia cinerea* in plums from Spain. Waterston (1947) records that the American brown rot of stone fruits, *Sclerotinia fructicola* has been intercepted in Bermuda on several occasions on peaches imported from North America but that it has failed to become established.

LOSSES DUE TO BROWN ROT DISEASES

The losses caused by the brown rot fungi are not easily assessable, particularly since the damage is so often to the trees themselves, destroying parts which might have borne fruit in future years. In Britain there are no statistics on the subject, but it is safe to say that brown rot of the fruit is to be found every year in any commercial plantation of top fruits, and the diseased fruit, fallen prematurely, rotting on the trees, rejected at picking time or during storage, constitutes a considerable part of the total crop. Again, in some years trees may be seen with almost the whole of their blossom destroyed, while branches and sometimes the upper parts of stems in cordon trees are killed by brown rot cankers. On some varieties of apples (e.g. Lord Derby) blossom wilt followed by cankers on the branches may become so destructive as to necessitate cutting back and top-grafting with another variety. In neglected orchards, blossom wilt and twig blight on acid cherries often destroy a large number of the young twigs every year, and standard trees particularly are often conspicuous by reason of their numerous dead bare branches. Losses which have resulted from blossom wilt on apple trees in the south of England are referred to on p. 65.

Abroad, losses from brown rot diseases are on a similar scale. On the Continent, rotting of fruit and blossom wilt are rife. In Germany, rotting of apples and blossom wilt in acid cherries are responsible for much loss every year. In the Rhône valley, brown rot of apricots not only reduces the crop but causes severe damage to the trees, while in the Crimea, the cultivation of the apricot, once a thriving industry, has almost been wiped out by brown rot.

In France, Gaudineau (?1948) announced that losses in peaches in 1945 amounted to 75 per cent for the varieties Mayflower and Charles Ingouff, 50 per cent for Amsden and 80 per cent for Triumph ; Chifflot (1921) estimated that apricot fruit yields in 1913, 1915 and 1916 were reduced by 80–90 per cent, and Weber (1926) found that 60 per cent of pears may be spoiled by brown rot in Denmark.

In America, approximate figures of losses are available especially with reference to brown rot of peaches and they again illustrate the destructive nature of the disease. In 1889, before control measures became general, E. F. Smith recorded that the brown rot fungus was more common and far more destructive than any other observed on the peach in this country. In rainy weather it frequently destroyed from one-half to three-fourths of the crop, in some cases the entire crop. Sometimes the greater part of entire varieties representing thousands of dollars were lost in three or four days and the loss to Georgia growers was sometimes as much as two-thirds of the whole crop. Hundreds of orchards in the upper portion of the Chesapeake peninsula produced no peaches whatever.

Stevens and Hall (1921) discuss losses from brown rot diseases. Among other facts, they mention 50 per cent loss in cherries as reported from New York and 25 per cent from Missouri in a single year. With reference to peach, they write that the disease ranges over the entire peach-growing territory but is usually most severe in the South. In some years the loss has amounted to more than 50 per cent of the crop or an aggregate annual loss of 5 million dollars. The estimated damage in Ohio alone in one year was a quarter of a million dollars. In one year in Pennsylvania twenty carloads were lost.

Roberts and Dunegan (1932), describing peach brown rot, write that in seasons of high rainfall they frequently saw the entire crop of individual orchards destroyed by the disease and the crop of whole sections reduced by as much as 75 per cent. Also, the plant disease survey of the Bureau of Plant Industry, United States Department of Agriculture, reported that in 1924 brown rot reduced the yield of peaches in the United States by 2,392,000 bushels, and in 1925 by 692,000 bushels. With reference to blossom blight they record having seen orchards in which 95 per cent of the blossoms had been killed by this phase of the disease.

Rudolph (1925) writing of blossom blight of apricots, states that unquestionably the loss in fruit, to say nothing of the great cost of fighting the disease, has amounted to hundreds of thousands of dollars annually in the past. In 1921 in one nine-acre orchard of large Blenheim apricot trees only one ton of fruit was picked whereas before Monilia blossom blight had invaded this orchard it regularly produced upwards of 90 tons a year.

Sharvelle and Teller (1950) mention 80 per cent fruit infection of Sapa cherry-plum hybrid variety on unsprayed trees. On Montmorency cherries, Petersen and Cation (1949) found 40–60 per cent of the flowers injured, and on sweet cherries 10–100 per cent varying with the season, locality and amount of inoculum present.

From records obtained from inspection certificates of the U.S. Bureau of Agricultural Economics, Brooks (1933) found that in a total of 7,846 cars of peaches inspected during the seven years (1922–28), 2,245 cars had 5 per cent or more affected with brown rot and some of the cars showed 20 per cent or more.

Bratley and Wiant (1950) write that at terminal markets brown rot is the chief cause of loss in all stone fruits. Fruit which appears sound when picked and packed may show high percentages of infected fruit after a three-day transit period.

With reference to brown rot of peaches and plums caused by *S. fructicola* in the delta of the Parana in Argentina, Fresa (1945) estimated that in years of severe attack, as much as 60 per cent of the crop may be destroyed.

In Australia, also, the brown rot diseases are very destructive to stone fruits in seasons favourable for infection. Darnell-Smith and Mackinnon (1915) report severe losses in cherries and nectarines. More recently Fish (1927) recorded that brown rot was the most destructive of all diseases of the peach in Victoria. In epidemics in 1918 and since, growers have lost entire crops. Harrison (1928), too, reports severe losses in certain seasons. Should favourable weather conditions occur early enough, brown rot can totally destroy the apricot crop. In 1919–20 approximately 500 bushel cases were destroyed in one orchard alone. The experience in the coastal districts of New South Wales is that the nectarine crop is very susceptible to brown rot damage. In 1927–28 in an orchard in which Cardinal nectarines were attacked by brown rot just as the fruit was maturing, not only was the whole crop destroyed, but the trees were so bady twig-blighted as to give the impression of having been " fired ". Numerous instances of severe losses of peaches have been recorded in both New South Wales and Victoria. In 1927–28 heavy losses occurred. In two orchards 12-year-old trees of Brigg's May and Hale's Early lost 80 per cent of their heavy crop to brown rot. These trees were so badly blighted as to necessitate cutting back to foundation leaders. There were many instances in which the whole crop of several varieties of plum was destroyed. Sometimes while losses in the orchard were moderate, fruit was destroyed before reaching the markets or before being consumed.

Other Australian observers mention 75 per cent loss of peaches in one locality of Victoria (Laidlaw & Brittlebank, 1922) ; 70–80 per cent loss in individual orchards of Phillips variety of canning peach in New South Wales (Noble *et al.*, 1937) ; while at a Conference of Australian plant pathologists held in 1949 it was agreed that an average yearly loss in stone-fruit production of 10–15 per cent due to brown rot would be a conservative figure (Pont, 1950).

In Tasmania brown rot is stated to be responsible for loss of half the crop in seasons favourable for the disease (Wade, 1951).

In New Zealand, Cunningham (1925*b*) states that brown rot is the most serious of all fungus diseases occurring on stone-fruits. With the exception of Central Otago (excluding Roxburgh), where it is unknown, it is common throughout New Zealand, occurring on apricots, cherries, nectarines, peaches and plums.

It is to be remarked that many of the losses reported above are exceptional, and such outbreaks occur only when the conditions are particularly favourable for infection. The losses from these diseases vary greatly from one year to another, yet every year the world's fruit crop is seriously diminished by brown rot. In some years, in certain localities, the effect may be almost negligible, but the fact remains that in others the results may be disastrous. The insidious nature of the brown rot diseases may be seen in this fact, that even in seasons unfavourable for the fungi there is enough infection to enable the organisms to survive, and there is then sufficient infective material on the trees to cause epidemic outbreaks when the environmental conditions favour the development of the fungi and the dispersal of their spores.

CLASSIFICATION AND NOMENCLATURE OF THE BROWN ROT AND RELATED FUNGI

Classification and Generic Names

The brown rot and related fungi have been subjected to many changes in classification and nomenclature. The earlier names *Oidium* and *Monilia* for the imperfect forms are mentioned above. When the ascigerous stages were found there was still much controversy over the genus in which they should be placed.

In Europe it was customary for some years to follow Boudier (1907) in recognizing the genus *Sclerotinia* with two sub-genera *Eusclerotinia* and *Stromatinia.* In the former the fructifications (apothecia) arise from a dense, compacted, black mass of fungal cells called a sclerotium, which develops in the stem or on the leaves of the host plant. In *Stromatinia* the fungus infects the fruit, and the mycelium and disorganized host cells together form a " stroma " which takes the shape of the mummy fruit ; the apothecia develop from this stroma. This is what occurs in the brown rot fungi. *Stromatinia* as the generic name for the brown rot fungi has been used by Ritzema Bos (1904) and by Delacroix and Maublanc (1909).

Honey (1928, 1936) proposed a new genus *Monilinia* to include the moniliform forms which show a genetic relationship to the genus *Sclerotinia*, with *Monilinia fructicola* as the type form.

The scope of recent papers on the classification of the Discomycetes to show the position of the brown rot fungi may not be out of place here. Using Martin's key (Ainsworth & Bisby, 1950), the ascigerous forms come in the ascomycetous order Helotiales (Discomycetes with inoperculate asci provided with a definite pore) and in the family Helotiaceae (with a periderm of elongate thin-walled and bright coloured hyphae arranged in parallel strands). Nannfeldt (1932), has a sub-family Ciborioideae (brownish apothecia often arising from a sclerotium or stroma and with conidial forms *Monilia, Botrytis* or none) containing the three genera:

Ciboria	Neither sclerotia nor pseudosclerotia present; macroconidia unknown.
Sclerotinia	Typical sclerotia: macroconidia, when found, of the Botrytis type.
Monilinia	With pseudosclerotia: macroconidia of the Monilia type.

Whetzel (1945) recognised a family Sclerotiniaceae to include forms producing sclerotia of various types. Those forms with Monilia as the imperfect stage produce a hollow sphaeroid sclerotium, and in his key Whetzel retains Honey's genus *Monilinia* for these forms. He lists 23 species of *Monilinia*.

More recently still Buchwald (1949) published a comprehensive review of the nomenclature of the Sclerotiniaceae with a synopsis of the genera, subgenera and sections. He retains Honey's *Monilinia* as a genus with the two sections:

(*a*) Disjunctors absent.	1. Sect. Junctoria (Eumoniliaceae).
(*b*) Disjunctors present.	2. Sect. Disjunctoria.

The brown rot fungi proper come under (*a*); the related fungi, with disjunctors between the conidia of the Monilia stage (Fig. 4) under (*b*).

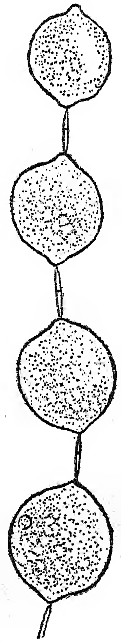


FIG. 4. Conidia of the Monilia Stage of *Sclerotinia cydoniæ*, showing disjunctors [× 1000]

The question arises as to the generic name which should be applied to the brown rot fungi, for unanimity in use would have obvious advantages. The choice now rests between *Sclerotinia* and *Monilinia*. The latter is frequently now used especially in American publications though in others the generic name *Sclerotinia* still appears. Honey's genus comprises a natural group of fungi and this might justify its general adoption. On the other hand Sclerotinia has become almost a popular name for the brown rot fungi; it has been used in hundreds of papers, past and recent, and its retention is desirable unless nomenclatural demands are insuperable. A suggestion which might meet the case is that *Monilinia* should be adopted as a sub-genus of *Sclerotinia* and in keys should be designated as such. Sometimes the Monilia forms, when grown in pure cultures, produce bodies which appear to be true sclerotia. The present writer has seen them in agar culture plates and they have been mentioned by others. This would suggest that the distinction between *Sclerotinia* proper and the *Stromatinia* forms is not sharply defined.

The name *Sclerotinia* is therefore retained in the general text of the present bulletin for the perfect stage of the brown rot fungi though in references to works by authors who use *Monilinia* or *Stromatinia* those names will generally be given, as being of some historical interest.

Biological Classification

From his observations and inoculation experiments Schellenberg (1923) concluded that the species of *Sclerotinia* on the Prunoideae and Pomaceae host plants could be put into three groups, viz.:

Forms not specialized

S. fructigena *S. cinerea* *S. laxa*	on all Pomaceae and Prunoideae and also on grapes, strawberries, and *Vaccinium* fruits

Specialized forms on Prunoideae

S. cerasi on *Prunus cerasi*
S. linhartiana on *P. padus*
S. pruni-spinosae on *P. spinosae*

Specialized forms on Pomaceae

S. aucupariae on *Sorbus aucuparia*
S. ariae on *Sorbus aria*
S. mespili on *Mespilus germanica*
S. crataegi on *Crataegus oxyacantha* and *C. monogyna*
S. cydoniae on *Cydonia vulgaris*

Nomenclature of the Species

Until comparatively recently the name *Sclerotinia* (*Monilia*) *fructigena* was used indiscriminately for any brown rot fungus by many workers who considered that the differences recorded were not sufficiently defined for more than one species to be recognized. More exact observations have indicated however that the common brown rot fungi comprise what are now considered to be three species.

The nomenclature of these three fungi has been the subject of much controversy. It was carefully studied by Harrison (1933) and although the position is not altogether unassailable, his conclusions give what is perhaps the best interpretation of the situation derivable from our present knowledge. He presents reasons for retaining the name *Sclerotinia fructigena*, only for the common fruit rotting fungus of Europe with buff-coloured Monilia fructifications: *S. laxa* for the fungus with the grey Monilia stage in Europe: and *S. fructicola* for the common brown rot fungus of North America. This nomenclature is used in the descriptions of the causative agents of the diseases described in this bulletin.

The argument is briefly this: Persoon's *Monilia fructigena* was definitely correlated with its Sclerotinia stage by Aderhold and Ruhland (1905), hence the name *Sclerotinia fructigena* Aderh. & Ruhl. is valid.

Monilia laxa and *M. cinerea* are morphologically indistinguishable and it is doubtful whether there is any biological difference between them. Again, Aderhold and Ruhland were the first to describe the perfect stage, giving it the name *Sclerotinia laxa;* hence this is considered to be the correct name for the fungus associated with blossom wilts in Europe. As inoculation experiments with strains taken from plum and cherry have failed to produce blossom wilt of apple trees, the apple blossom wilt fungus is considered to be a biologic form of *S. laxa,* and the combination *Sclerotinia laxa* Aderh. & Ruhl. forma *mali* (Wormald) Harrison has been proposed for it.

Killian (1921, 1926), as a result of studying various strains of *S. cinerea* in culture, considered that on cherries there were two forms, and these he designated *S. cinerea* f. *avium* on sweet cherries, and *S. cinerea* f. *cerasi* on acid cherries. Whether these forms are essentially anything other than cultural strains is doubtful. Killian himself found that each form could infect the other host, and inoculation experiments carried out at East Malling have shown that blossom wilt of Morello cherries can be induced by *S. cinerea* obtained from sweet cherry.

The brown rot fungus generally distributed throughout the fruit-growing regions of North America was at first considered to be *S. fructigena*, and this name was adopted by Norton (1902) who first connected the American brown rot *Monilia* with its ascigerous stage; later it was found to be more closely related to *S. cinerea* (Matheny, 1913), so this name was adopted by a number of workers. A more careful comparison of the American fungus with *S. cinerea* [*S. laxa*] by Wormald (1920*a*) in England, and Norton and Ezekiel (1924) in America, showed that the two were not identical and the name *Sclerotinia americana* (Wormald) Norton & Ezekiel was proposed for the American fungus. Pollock (1909), however, had pointed out that an ascigerous fungus described by Winter in 1883 as *Ciboria fructicola* (later changed to *Sclerotinia fructicola* by Saccardo on the authority of Rehm) was probably the common brown rot fungus of North America, and more recent studies have shown that it is indistinguishable from the *Sclerotinia* known to be genetically connected with the American brown rot *Monilia*. Winter's fungus, however, was never connected with a Monilia stage, and his description does not distinguish between the common American fungus and *Sclerotinia laxa* which also occurs in some of the States. Certain American workers, in particular Roberts and Dunegan (1927*b*), consider therefore that the brown rot fungus generally distributed in the fruit-growing regions of North America should be known as *Sclerotinia fructicola* (Wint.) Rehm, and their point of view is shared by Harrison (1933).

Fungi related to the Brown Rot Fungi

Other Sclerotinias which infect rosaceous plants and have a Monilia as the conidial stage have been described in Europe, in North America and in Japan.

Takahashi (1911) described a cherry disease caused by *Sclerotinia kusanoi* and a blossom blight of the apple. The symptoms of the latter bear some resemblance to those of blossom wilt of apples in Britain, but the conidia of the fungus are stated to be provided with disjunctors, which thus distinguish it from any form of *S. laxa*. Susa (1934) published (in Japanese) an account of an apple flower rot caused by *Sclerotinia malicora* Miura, with illustrations showing blossom wilt and a leaf with typical leaf blotch symptoms, while Shima (1936) described in great detail, in English, a disease of apple trees in Japan caused by *S. mali* Tak., a fungus which attacks leaves, blossoms, young fruits and shoots.

Fungi related to those causing brown rot are found in Europe (see particularly Schellenberg, 1923, and Eriksson, 1930), and three of these occur in Britain. They usually produce a leaf blotch, a form of disease caused by certain species of *Sclerotinia* that have a Monilia stage characterized by possessing disjunctors, i.e. narrow barren separating cells between the conidia (Fig. 4). In Britain leaf blotch occurs on quince, medlar and hawthorn. The disease is first seen as a dark, almost black, blotch which increases in size until the whole leaf is involved (Plate VA). Meanwhile the fungus appears on the upper surface of the lamina as a grey mould, chiefly along the midrib and main veins; infected leaves emit a sweetish odour. Direct infection of the flowers has not been observed in Britain, but wilting of the flowering shoots of quince and medlar may take place by invasion of the shoots from the infected leaves (Wormald, 1920*a*, 1926).

The Sclerotinia stage of the quince fungus *S. cydoniae,* has not been observed in this country, but it has been found on the Continent. That of *S. mespili* has not been found in Britain under natural conditions in the orchard, but it was obtained by collecting young mummified fruits from the ground in spring and keeping them in soil in a pot ; the perfect stage appeared in the following spring (Wormald, 1922*b*).

A leaf blotch caused by *S. crataegi* was found on hawthorn in Kent in 1926 (Wormald, 1928*c*) and since then it occurred sporadically until 1937, when outbreaks occurred in many parts of the country ; it was particularly severe on hedgerows round Cambridge (Dowson & Dillon Weston, 1937).

Quince leaf blotch may sometimes be severe and on one occasion more than a hundred dead shoots were counted on one tree. From an economic point of view this disease and those on medlar and hawthorn are not important, but they are mentioned here because of the close relation which the fungi causing these diseases bear to the brown rot fungi proper, and also because the symptoms rather suggest those of shoot wilt of plums, and the grower may suspect medlars and quinces to be a source of brown rot infection for other fruit trees. These leaf blotch Sclerotinias, however, are not known to attack other fruit trees.

Early observations on disjunctor Sclerotinias were made by Woronin. In 1888 he gave an account, with striking coloured illustrations, of apothecia, of Sclerotinias on *Vaccinium* spp., and, in 1895, of *S. padi* (on *Prunus padus*) and *Sclerotinia aucuparia* (on *Pyrus aucuparia*). These observations are interesting not only as calling attention to the disjunctors in the Monilia chains of these fungi but also because they induced Woronin (1900) to place *Monilia fructigena* and *M. cinerea* in the genus *Sclerotinia* before the perfect stage of these two fungi had been discovered.

Other disjunctor Sclerotinias have been discovered from time to time, some of them being of horticultural interest. On rosaceous hosts, *Monilinia amelanchieris* has been recorded on *Amelanchier* spp. (Honey, 1942), *Monilinia seaveri* found first on a wild cherry, *Prunus serotina,* and more recently on the Morello cherry (Dunegan, 1940, 1947), and *Sclerotinia chaenomelis* on *Cydonia* (*Chaenomeles*) *japonica* in Switzerland (Fischer, 1930).

On other cultivated hosts may be mentioned: *Sclerotinia oxycocci* on cranberry (Shear *et al.,* 1931), *Monilinia vaccinii-corymbosi,* the cause of mummy berry of the cultivated blueberry (Bailey, 1948), *Sclerotinia camelliae,* producing a flower blight of camellias (Hansen & Thomas, 1940), *Ovulinia azaleae,* the azalea petal blight fungus (Honey, 1940 ; Brierley, 1950) ; this disease has been found also in Switzerland (Terrier, 1950).

Other fungi to which reference may be made as having some bearing on the brown rot problem are:

Monilia (*Moniliopsis*) *folliicola,* found originally by Voronichin on leaves of *Pyrus communis* on the Caucasian Coast of the Black Sea and described by Siemaszko (1924) who found it in the same region and in Poland.

Monilia roreri, associated with a serious rot of cacao pods in certain regions of South America. It was at first supposed to be *Monilia cinerea* (Ciferri, 1928), but is now considered to be distinct (Ciferri & Parodi, 1933). (The distribution of *M. roreri* is shown in Distribution Map No. 13 of the Commonwealth Mycological Institute, p. 10.)

Sclerotinia sclerotiorum in California causes a green rot of apricots (R. E. Smith, 1931), with a complete or partial destruction of the young fruit ; this disease has been recorded from Queensland (Morwood, 1931).

Sclerotinia aestivalis Pollock (syn. *Ciboria aestivalis* (Pollock) Whetzel) was found by Harrison (1935*b*) in New South Wales; the apothecia were very abundant on mummified apples, apricots, peaches, pears, plums and quinces, and in culture. He considered it to be practically a saprophyte but it caused a slow rot of apples under favourable conditions.

Three species of *Lambertella*, a genus of apothecial brown-spored fungi have been found on rosaceous fruits (Whetzel, 1945). *L. corni-maris* collected originally in 1917 by von Höhnel (1918) in Austria on mummified fruits of the cornelian cherry (*Cornus mas*), but more recently by Harrison on mummified fruits of apple and pear in Switzerland and South Germany in 1931. Under laboratory conditions it attacked apple, pear, plum, quince and other fruit (Harrison & El-Helaly, 1935). *Lambertella jasmini* was found first on mummified jasmine berries but Whetzel (*loc. cit.* p. 40) mentions its occurrence on loquat, *Eriobotrya japonica* as probably accidental rather than habitual, and English (1945) isolated a fungus resembling it from rotting sweet cherries. *L. pruni* has been found on sweet cherry (*Prunus avium*) mummies (Whetzel, *loc. cit.* p. 42).

MORPHOLOGY AND CYTOLOGY OF THE BROWN ROT FUNGI

In the life history of the brown rot fungi there are two stages each with its particular type of fructifications and spores. The more familiar stage is that found in summer on infected fruits, flowers and twigs, in the form of dense velvety tufts (*sporodochia*) of upright or somewhat spreading conidiophores terminating in branched chains of ellipsoid, ovoid or lemon-shaped, one-celled, colourless conidia. This, the imperfect stage in the life history, belongs to the form-genus *Monilia* (Fig. 5).

The perfect stage is *Sclerotinia*. This fruit body in the brown rot fungi is a stalked brown cup (*apothecium*) growing out of mummified fruits which have overwintered on the ground. Few or many apothecia may develop from a single mummy: Norton *et al.* (1923) counted as many as 675 apothecial primordia on one mummified peach, but stated that rarely over 25 fully developed apothecia appear on one mummy under natural conditions. The stalk (*stipe*) varies considerably in length, depending on the length necessary to bring the cup to the surface of the soil if the mummified fruit is buried. The fully expanded cup is usually less than 1 cm. in diameter, but large specimens of *S. fructicola* may be 1·5 cm. across. The mature apothecium may be almost flat and the edges may be recurved. The inside of the cup is lined with a layer of parallel elongated cells (*asci*), interspersed

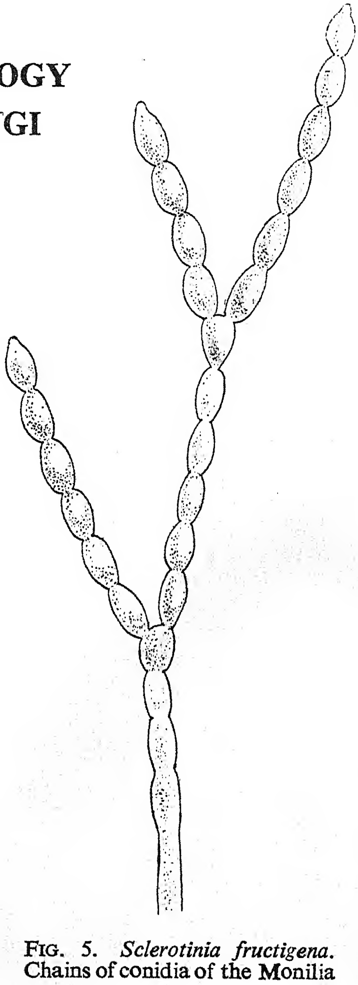


FIG. 5. *Sclerotinia fructigena*. Chains of conidia of the Monilia stage.

with simple or feebly branched hyphae—the paraphyses—which are about the same length as the asci (Plate VIA).

The ascus is a cylindrical sac containing eight ellipsoid, one-celled, hyaline ascospores (Plate I). When the asci are ripe the spores are ejected. In *S. laxa,* the ejection of the spores has been found to be accompanied by a rapid elongation of the ascus and the assembling of its eight spores in the apical region shortly before they are finally shot out at the apex (Wormald, 1921).

In Europe, the apothecia of brown rot fungi have rarely been found. Aderhold and Ruhland (1905) described them first for *S. fructigena* and *S. laxa.* Those of *S. fructigena* have since been found by Manaresi (1920) in Italy and by Solkina (1931) in Russia; they have not yet been seen in Britain. Fructifications which were probably those of *S. laxa* were found on cherry by Westerdijk (1912) in Holland, and in 1920 they were obtained on plums at Wye, Kent (Wormald, 1921). The ascigerous fructifications of *S. fructicola* on the other hand are common in the United States, Canada, Australia and New Zealand. Harrison (1935*a*) critically discusses these records in some detail and describes some of his own observations on the brown rot apothecia.

As a rule the apothecia develop from the mummies during the second spring after infection. The apothecia of *S. laxa* were obtained at Wye by collecting, during the winter, mummies of plums infected during the summer, placing them in pots in January and leaving them exposed in the open all through that year and the following winter; the apothecia appeared in March (Plate VB).

With *S. fructicola,* Ezekiel (1921) observed that under natural conditions apothecia may develop in the spring following inoculation. Cold is probably a factor influencing the production of apothecia, as apothecia were produced in 25 weeks from chilled mummies. Burying mummies below the surface of the ground inhibits the production of apothecia. Pollock (1918) found that plum mummies 10 years old were still capable of developing apothecia (Plate VC, D).

The form of the apothecium is similar in all three species; the asci and ascospores, too, are very much alike, but one characteristic of *S. fructigena* is that the ascospores are pointed, rather than rounded, at the ends.

There are no constant morphological differences in the apothecial and the conidial stages of *S. laxa* and *S. fructicola,* but differences in habit of growth in pure cultures and in the mode of germination of the conidia of the two fungi have been recorded, and their relations to their host plants appear to be not identical; *S. laxa* is chiefly a blossom wilt fungus though it also causes fruit rot, while *S. fructicola* is mostly a fruit-rot fungus, but it, too, can infect flowers.

The general development of the apothecia of *S. fructicola* in relation to environmental conditions has been studied by Norton *et al.* (1923), who find that, after slow development for more than a month, the apothecia mature at the time peaches bloom.

In the germination of the conidia on culture plates, the germ tube of *S. laxa* soon becomes strongly curved (geniculate) and branches; that of

S. fructicola is straighter and less branched in the early stages. In *S. fructigena* the germ tube grows out even further than in *S. fructicola* before it branches (Wormald, 1920*a*; Ezekiel, 1924*b*) (Plate IV).

In pure cultures these fungi not only produce ordinary conidia, but they also develop microconidia; these are spherical bodies 2–4·5 in diameter (mostly about 3μ), produced in chains from the apices of flask-shaped cells. Their function has been open to conjecture for a long time. Humphrey (1893) claimed to have seen them germinate and give rise to the Monilia form in culture. Most investigators, however, have failed to germinate them, and it was generally considered that they were not reproductive bodies. When, however, Drayton (1932) announced that he was able to induce the development of apothecia in *Sclerotinia gladioli* by placing microconidia of one thallus on certain structures which develop on another thallus, it seemed not unlikely that the microconidia of the brown rot fungi might have a similar "spermatization" function. This has not yet been proved experimentally, but the structure and development of the ascogenous hyphae of *S. fructicola* as illustrated by Heuberger (1934) (Plates II, III), and the segregation in the asci of spores with different cultural potentialities as mentioned by Sharvelle and Chen (1943), and described in some detail by Thind and Keitt (1949), do suggest that a sexual process is involved in the development of the apothecium, and that the microconidia are spermatia.

The development of apothecia from fruits inoculated with single spore cultures of *S. fructicola* indicates that this fungus is homothallic (Ezekiel, 1924*b*). It seems possible that this is correlated with the great frequency with which the apothecia are found in those regions where *S. fructicola* abounds, and suggests that *S. laxa* and *S. fructigena,* which produce apothecia rarely, are heterothallic.

The characters of the reproductive organs of the three brown rot fungi obtained by correlating the results of various observers are as follows (dimensions of asci and spores are given in μ):

	Apothecial Stage		**Conidial Stage**	
	Asci	*Ascospores*	*Sporodochia*	*Size of Conidia*
S. fructigena	112–180 × 9–12	9–12·5 × 5–6·8	Buff-coloured about ⅙–⅛ in. in diameter	12–34 × 9–15 av. about 22 × 13
S. laxa	121–188 × 7·5–11·8	7–19 × 4·5–8·5	Grey: variable in size, but usually about half the diameter of those of *S. fructigena*	Winter conidia 5–19 × 4–12 av. about 11·5×8. Summer conidia 8–23 × 7–16 av. about 19 × 13·5
S. fructicola	102–215 × 6–13	6–15 × 4–8·2	As in *S. laxa*	10–27 × 7–17

The dimensions of the asci in *S. laxa* are those taken while the spores were still in one row (monostichous); just before dehiscence. when the spores assemble towards the apex in two rows (distichous) the asci measure 162–226×10–13μ.

In *S. laxa* the winter conidia are those which develop in winter and early spring on organs in which the fungus has overwintered. The summer conidia are those which develop on newly infected flowers, fruit and leaves, during late spring and in summer. It will be seen that the summer conidia are about one and a half times the length of the winter conidia.

Harrison (1935*a*), from a comparative examination of *S. laxa* and *S. fructicola,* considers their apothecia to be indistinguishable morphologically, but that the differences in the imperfect stage warrant their separation. The cytology and development of the asci in *S. fructicola* have been studied by Heuberger (1934).

CONDITIONS CONDUCIVE TO INFECTION

In the brown rot diseases, as with other plant diseases caused by fungi, the primary factors necessary for infection to be initiated, and for the fungus mycelium to attack the host tissues, are:

The inherited susceptibility of the host plant;

The presence of the fungus;

Conditions favourable for the development of the fungus and for its penetration into the host plant;

A physiological or morphological condition in the host plant allowing of infection.

Inherited Susceptibility and Resistance

The primary condition for infection is the juxtaposition of a susceptible host plant and the parasite. Infection by the three fungi under consideration is confined almost exclusively to species of the suborders Pomoideae and Prunoideae of the family Rosaceae though other species have been infected by artificial inoculation and natural infection of grapes has been recorded.

The degree of susceptibility to brown rot varies widely among related species and among varieties of particular species. In cherries the outstanding example of specific difference is that between the very susceptible acid cherries (*Prunus cerasus*) and the resistant sweet cherries (*P. avium*). In the former the extreme susceptibility of the Morello cherry has been noted in Britain (see p. 74), and in Europe it has been mentioned by many observers ; early accounts have already been referred to and further observations have been made by Köck (1910) and others.

In relation to wild species of *Malus* and *Pyrus,* Kovalev (1940) concluded that there was a correlation between the degree of immunity of a species and its geographical origin—that species of *Malus* from the humid regions of Eastern Asia are definitely resistant to Monilia fruit rot. He explains this by assuming that in those regions there have been selected and definitely fixed, during the course of thousands of years, a number of forms resistant to the chief diseases, whereas under the relatively arid conditions of Western China and Soviet Central Asia selection for resistance to diseases of plants of humid climates could not take place.

Varietal susceptibility to brown rot has frequently been observed and commented on. M. J. Berkeley (1864) remarked that the Moor Park apricot was more subject to attack than other varieties ; no infection was observed on the Roman apricot and on the Breda variety he saw only a single infection. Of the varieties of apricot grown in the Rhône valley Chifflot (1921) states that Luizet is the most resistant and is followed in order of resistance by Abricot d'Ampins, Blanc rosé, Paviot and Poizat.

Varietal differences in susceptibility have often been recognized in cherries and plums. On the Continent, Köck (1910) records observations on 27 varieties of cherries; Grosse Lange Lotkirsche was severely damaged while the variety Beste Werdersche was not infected although its twigs extended among those of the Lotkirsche. It was suggested however that the difference in degree of infection might have been due to difference in time of flowering as related to the weather conditions at flowering time. Elssmann (1939) tested acid cherry varieties in Bavaria and found that one variety proved to be immune to *Monilia cinerea*, that others developed gummosis but no wilting, and that severe wilting was never accompanied by gummosis, and *vice versa*. Mittmann-Maier (1940*a*) confirmed that in acid cherries gumming is an indication of resistance. Among sweet cherries recently raised in our own country it was noticed by Hart (1947) that the variety Merton Heart is markedly resistant to blossom wilt (*Sclerotinia laxa*).

Varietal susceptibility to *Sclerotinia fructigena* on apples was examined by Katser (1933) who found that Jonathan, Grüner Fürster, and Rote Jungfer varieties were uniformly resistant even under conditions favouring the fungus, while Kaiser Alexander and others were consistently susceptible. Mittmann-Maier (1940*b*) tested apple and pear varieties by artificial inoculations, and lists a number of varieties which proved to be resistant.

American workers, too, have made detailed examination of varietal susceptibility to brown rot with reference to *Sclerotinia fructicola* on stone fruits—apricots, cherries, peaches, and plums.

Very susceptible varieties of apricot are grown in central California with others that are resistant. Some varieties transmit resistance to their progenies but Royal transmits susceptibility, and hybrids of the susceptible St. Ambroise are among the most resistant, regardless of the other parent (Hesse, 1939). The genetics of resistance to brown rot in apricots would thus appear to be a complex problem.

Schmidt Bigarreau cherries in New York State show extreme susceptibility to brown rot (Gloyer, 1928).

Varietal reaction to brown rot on plums in Indiana was noted by Burkholder (1943), who remarks that the damson group, especially French and Shropshire, rarely need more than two lime sulphur sprays during July and August to protect them against infection, very little of which will occur in dry seasons even without treatment. Other resistant varieties also are listed, but some varieties, on which *S. fructicola* occurs in a very virulent form, are late ripening and require a supplementary sulphur spray in September.

With reference to varietal susceptibility in peaches, Roberts and Dunegan (1932), wrote that the modern commercial varieties of the peach showed considerable resistance to brown rot in normal years. One of the reasons why they had supplanted the older and often better-flavoured varieties was that they were less susceptible to brown rot in the orchard, in transit, in the market, and in the home. Practically all modern commercial varieties were more resistant to the blossom blight phase of the disease than were the older ones, but all might be quite susceptible under optimum conditions for infection. It is to be noted however that since that time the control of brown rot in peaches still remains a major fruit-growing problem in the United States and Canada, as shown by the numerous papers published on the subject.

Hurt (1937) mentions J. H. Hale and Red Bird varieties as being most susceptible to peach blossom blight in Virginia.

Some consideration must be given to the possibility of biological forms of the fungi with different infection potentialities on different hosts. Wormald (1919, 1920*a*) described *S. laxa* f. *mali*; the typical form of *S. laxa* is almost confined to drupaceous species of fruit trees, but forma *mali* attacks apple flowers causing serious blossom wilt. Other biological forms have been mentioned. Thus Killian (1917, 1921, 1926) describes two forms of *S. laxa* as *S. cinerea cerasi* on acid cherries and *S. cinerea avium* on sweet cherries.

The question of biologic forms of *Monilia cinerea* on stone fruits was examined by Staehelin (1946) by cross inoculations. He infected cherry flowers with conidia from peach, cherry, plum and Mirabella, and Morello flowers with conidia from peach, apricot, plum and Mirabella, and concluded that the species *Monilia cinerea* cannot be subdivided into different races. With apricot, however, infection was not so successful and in 18 tests, with about 400 flowers and using conidia from various sources, he obtained infection on only one flower.

Factors relating to the Presence of the Parasite

DISSEMINATION. The fungus may be already present in the neighbourhood of a susceptible host particularly on previously infected plants or plant parts, or it may come in from elsewhere.

The brown rot conidia probably travel considerable distances in the air, as shown by the occurrence of outbreaks of brown rot in new regions, in new plantations, or on trees that have not previously shown infection. The attack on any particular tree is worst, however, when sources of infection are already present on the tree. In blossom wilt of apple trees and of Morello cherries it has been noticed repeatedly that the newly infected flowers are mostly in close proximity to cankers, or old infected spurs and twigs (Fig. 6). When a relatively high number of dead flower trusses are seen in one part of a tree, the source of infection can almost invariably be found near them, usually at a little distance above the dead trusses.

That the conidia of the brown rot fungi travel through the air has been demonstrated by a number of observers. In our own country Horne (1933) by exposing culture plates in the air for short periods caught the conidia of *Monilia fructigena* in orchards at East Malling and at Swanley, Kent. In Germany, Bucksteeg (1939*b*) studied the dispersal of brown rot conidia in the air of the lower Elbe districts by the smeared glass slide method. Some conidia were found almost throughout the year but they were most numerous during April and again in June, particularly early June in acid cherry orchards, and late June or early July in pome fruit orchards. Wiesmann (1932) estimated that the conidia of *Monilia fructigena* on apple are detached by a wind velocity of 60 metres per second. Christoff (1934*b*) describes an apparatus for catching wind-borne spores for determining the date of first appearance each year of plant pathogens in Bulgarian orchards, and *Monilia cinerea* was one of the fungi caught.

In America, Cohen (1942), collecting *Monilia* [?*Sclerotinia fructicola*] conidia on petri dishes found that they were most numerous from June to September with the peak period in July. Wilson and Baker (1946*a*, 1946*b*), taking blossom wilt as a type of fungus disease spread by air-borne spores, determined that the observed spread is described to a good first approximation by an equation of the form $y=A/x^p$, where y is the ratio of the percentage of blossom infection in a vertical slice of susceptible tissue (blossom) at a horizontal distance from the source block to the percentage of blossom

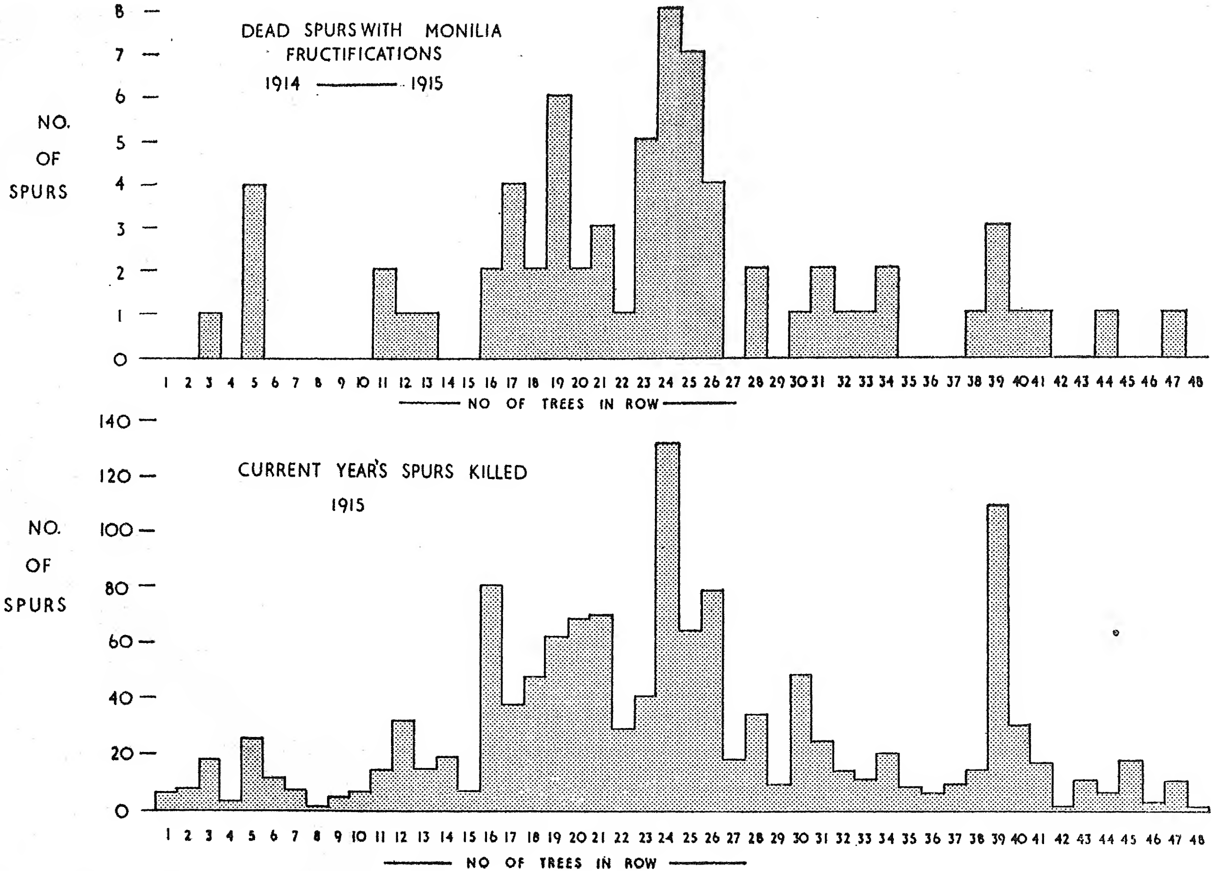


FIG. 6. Graph showing, in a row of apple trees, the relation between the number of conidia-bearing spurs killed the previous year and the number of infected inflorescences of the current year on the same trees.

infection in the source trees, A and p are constants depending on wind velocity and perhaps on other quantities to a lesser extent, and x is the horizontal distance from the centre of the nearest source trees.

In America, where apothecia of *Sclerotinia fructicola* are so common, ascospore discharge and dissemination have received careful consideration. According to Norton's (1902) observations, the spores are thrown off as the disc dries up. If fresh apothecia are kept for a time in a moist atmosphere and then exposed to a drier, or blown upon by currents of air or the breath, a distinct cloud of spores can be seen discharged which ascends several centimetres high and is wafted away by the slightest air current. When held in the sunlight the individual spores can apparently be seen with the naked eye. The discharge of spores can be several times repeated by additional drying, and after a rest still others may be given off. Not all the spores are ejaculated, as old dry individuals contain many asci intact and others with a few spores. He obtained almost pure, agar plate cultures by holding a sterile cover-glass with a drop of liquid for a moment over the discharging asci, dropping it into a tube of bouillon and then preparing the plates in the usual manner.

Roberts and Dunegan (1932) write that the ascospores are discharged by pressure within the ascus, and the simultaneous discharge of many asci from a single apothecium produces a dust or cloud easily discernible to the naked eye, and they describe an apparatus for observing their discharge. They found that the average height of spore discharge was 1·7 cm., the highest discharge observed being 4·3 cm. and the lowest 0·5 cm. They collected apothecia which produced clouds of spores at intervals for as long as seven days. Culture plates exposed for three minutes at a height of four feet above the ground each developed one colony.

Harrison (1935*a*), made observations on the same fungus in Australia, and observed clouds of ascospores being wafted, like thin smoke, through the air for a height of from 2 to 3 metres. He also remarks that the spores are so light that the initial forces of expulsion start an upward movement sufficient to enable them to reach the flowers of stone fruit trees, where infection may result. A definite correlation between apothecial production and the occurrence of blossom wilt and twig blight was established in several districts of New South Wales in 1932 and 1934.

Insects are not only instrumental in causing wounds allowing of infection, as mentioned below (p. 33), but they are suspected also of assisting in the dispersal of brown rot diseases by carrying the conidia on their bodies. In this connection, Joëssel (1932*a*) and Pohl (1939) mention hornets and wasps, Dillon Weston (1945) wasps and flies, and M. H. Moore (1950*b*) wasps. Possibly hive bees, too, aid in dispersing conidia ; they, together with wasps, are frequently seen on fallen, rotting, fruit, and even butterflies are sometimes attracted to rotting apples on the ground. Dillon Weston and Petherbridge (1933), during a study of apple and pear scab in East Anglia, found conidia of *Monilia cinerea* on the legs of the leaf-curling plum aphid (*Anuraphis helichrysi* Kelt.). This observation shows that brown rot may be spread by aphids and also offers partial explanation of the way in which tar oil washes indirectly reduce attacks of this fungus.

Other factors contributing to the dissemination of brown rot are of a more mechanical nature. That man may be an unconscious agent in distributing the disease on exported fruit is referred to elsewhere (p. 16). It is very probable, too, that nursery material of fruit trees also carry one or other of the brown rot fungi, otherwise it would be difficult to account for the presence of *Sclerotinia fructicola* in Australasia, which in the early days of fruit growing there, must have imported young trees, possibly mostly from the United States.

ENVIRONMENT. Conditions most favourable for the development of the fungi in the field are dull weather accompanied by frequent showers or mists, so that the atmosphere remains saturated, or nearly so, for long periods. Under such conditions the brown rot fungi produce and shed conidia rapidly. Mists at the time the trees are coming into flower are particularly conducive to blossom infection. Severe infection of unopened flowers of the Morello cherry have been observed to follow misty weather as the buds were bursting, and destructive outbreaks of brown rot on stone fruits, particularly apricots, in the coastal regions of the Black Sea, have been noted by Russian workers after frequent periods of sea mists.

Such conditions are not only directly favourable for the development of the fungi ; they are a disadvantage to the host plant, in that after the trees have come into bloom the flowers remain open, and are therefore liable to infection, for comparatively long periods, for cold, wet and dull weather hinders pollination since it checks the activity of pollinating insects.

Many observations on the correlation of weather conditions with brown rot infection have also been made with reference to *Sclerotinia fructicola* on drupaceous fruit trees in America and Australasia.

In California, Hunt (1923) noted very severe infection of apricot flowers after a wet spring and states that the brown rot fungus is unable to make its attack upon the blossom in the absence of moisture.

Weaver (1950) studied the effect of temperature and relative humidity in relation to infection of peach flowers by *S. fructicola.* Conidia germinate at 5–30°C., the optimum temperature being 15–25°C. Dusted on petals, stamens and stigmas of peach the conidia germinated on all organs at 100 per cent relative humidity, on only the stamens and stigmas at 96 per cent and only on the stigmas at 80 per cent. They infected blossoms in a saturated atmosphere within 18 hours at 10°C., 8–12 hours at 15°C., 11 hours at 20°C. and 8 hours at 25°C. Open blossoms are more liable to infection than unopened buds, but unopened blossoms were infected at high relative humidities. Infection occurred on exposed stamens and stigmas.

Injuries allowing Infection

Alternation of wet and dry periods (particularly a wet period following a drought) often results in the rupture of the skin of fruits as a result of the internal tissues growing at a greater rate than the skin. Such rupturing followed by brown rot infection is frequent in cherries when there is a rainy period round about the time of picking ; it is also common in plums and has been observed in apples. Maier (1942*b*) records serious brown rot infection (up to 85 per cent) in apricots following splits and cracks in the fruit ; he suggests that the splitting may be prevented by appropriate cultural operations. M. H. Moore (1932, 1933) found that pre-blossom spraying with Bordeaux mixture caused severe cracking on Cox's Orange Pippin fruit and that brown rot (*S. fructigena*) was commonly found on such fruits. Mechanical injuries from hail, or bruises caused by the fruit coming in contact with other parts of the tree in high winds, also allow of infection, as do also bruises and other injuries to the fruit during picking and marketing operations.

Primary infection of fruit by other fungi sometimes causes ruptures in the skin which serve as ports of entry for the brown rot fungi. Thus there is evidence that apple scab (*Venturia inaequalis*) lesions allow the entry of *Sclerotinia fructigena* (Grubb, 1921 ; Moore, 1932, 1950*b*, 1950*c*) (Plate VIB), and Ovtshinnikova (1937) noticed that on pear trees *S. fructigena* developed almost without exception in wounds or cracks caused by *Fusicladium pirinum.* Scab (*Cladosporium carpophilum*) on stone fruits is said to cause cracks at which infection by *Sclerotinia fructicola* arises (Rose *et al.*, 1937) in America, and Joëssel and Anrès (1932) mention that infection on apricots in the Rhône valley may take place through *Coryneum* [*Clasterosporium*] *carpophilum* lesions but that it is relatively rare.

The most frequent wounds allowing brown rot infection are those caused by insects, either during oviposition or when in search of food or moisture. In some relatively dry seasons brown rot of the fruit is common and this is probably related to the increased number of insects. Moreover in drought periods such biting insects as wasps frequently attack ripening fruit, producing injuries that allow infection.

Injuries caused by insects in their relation to brown rot infection have been noted and emphasized by many observers, and rightly so, for although they are so frequent they can to a large extent be prevented if attention is given to insect control, in contrast to the climatic conditions that favour infection and cannot be modified by cultural operations.

In our own country the codling moth is one of the chief offenders. Moore finds (1950*b*) that codling moth (*Cydia pomonella*) and scab were the chief wound agents functioning as precursors of apple brown rot and Osterwalder and Kessler (1934) mention codling moth as furthering brown rot infection in Switzerland.

That earwigs (*Forficula auricularia*) can produce injuries which allow of the infection of apples by *Sclerotinia fructigena* is shown by Croxall *et al.* (1951), who found the damage to be more noticeable in grass orchards than in arable plantations. Bands of sacking soaked in benzene hexachloride and placed round the trunks trapped many earwigs and there was less brown rot on the banded than on control (not banded) trees.

In Europe *Rhynchites bacchus* appears to be one of the chief insects to cause fruit injuries which admit brown rot. Joëssel (1932*b*) mentions this insect and also that the holes caused by the Mediterranean fruit fly (*Ceratitis capitata*) at oviposition allow infection by *S. laxa.* Kazanskii (1935) made a careful study in the Crimea of the relation between insect injury and brown rot infection of apricots, plums and apples. He states that *Rhynchites bacchus* is the chief agent in the dissemination of *Monilia,* that the red plum maggot (*Cydia funebrana*) is responsible for 50 per cent brown rot infection in plums but that *Hoplocampa testudinea* (the apple sawfly) and *H. flava* (plum sawfly) tend to render fruits attacked by them, immune from *Monilia* infection. Ovtshinnikova (1937) finds that brown rot of pome fruits mostly enters through injuries caused by *Rhynchites bacchus,* and a number of Tortricid larvae. Chaboussou (1945) says that *Rhynchites bacchus* occurs on all kinds of fruits but particularly on d'Ente prune and nectarine in the south-west of France.

Moore (1947, 1950*a*) finds the nut drop of cultivated hazel nuts is primarily due to the oviposition punctures of the Nut weevil *Balaninus nucum* and that *Sclerotinia fructigena* enters through the punctures and causes a rot.

In the United States the injuries caused by the curculio (*Conotrachelus nenuphar*) are widespread and their relation to infection of stone fruits is frequently mentioned (e.g. Scott & Quaintance, 1911). The oriental fruit moth (*Grapholitha molesta*) is another insect often associated with brown rot infection caused by *Sclerotinia fructicola* (Roberts & Dunegan, 1932). Scott and Ayres (1910) estimated that 93 per cent of the peaches with brown rot are infected through curculio wounds.

Rose *et al.* (1937) also mention curculio, oriental fruit moth and syneta leaf beetle (*Syneta albida*), and Daines (1942) estimated that in most years 90 per cent of the brown rot infection in peach orchards takes place through insect punctures, chiefly those of curculio and oriental fruit moth and that in some orchards Japanese beetles may become an important factor in increasing brown rot infection. The importance of insect control in reducing brown rot is emphasized by Marshall (1949) who finds that in Indiana effective control of plum curculio and oriental fruit moth automatically reduced brown rot to a very minor problem.

In New Zealand codling moth and the bronze beetle are suspected (Cunningham, 1925*a*) of allowing infection of pome fruits by *S. fructicola.*

Birds not only injure the fruit by pecking holes in it, allowing brown rot infection, but they probably carry the conidia about with them, and one case is recorded of birds transporting infected mummified fruit from one place to another. This was observed in Slovakia by Turček (1950) who obtained evidence that the pied woodpecker (*Dryobates maior pinetorum*) fed on mummified plums and transported them from one garden to another, and that as the fungus remains viable till the spring, this bird may be a factor in the dissemination of brown rot.

Factors modifying Susceptibility and Resistance

Factors which modify the susceptibility or resistance of fruit to brown rot have been investigated or noted by a number of workers. There is evidence that in apples the rootstock affects the susceptibility to infection by *Sclerotinia fructigena* of the fruit of the scion variety grafted on it. Thus fruit of trees of Cox's Orange Pippin on rootstocks M.XV, XIII and III showed less tendency to brown rot infection than those of trees on other stocks (Moore, 1932), and again, the rootstock may, at any rate indirectly, affect the incidence of brown rot on that variety, for on M.IX it is specially prone to russeting and cracking which produce ruptures in the skin allowing of brown rot infection.

The degree of ripeness has some effect on the readiness with which the fruit becomes infected. Cooley (1914) found that ripe or nearly mature plums could be easily infected by sowing a spore suspension of *S. fructicola* on the uninjured surface whereas unripe fruit usually required a wound for the fungus to enter. The decay of apples inoculated with *S. fructicola* is less rapid, however, on the blushed than on the green side (Lin, 1942) ; the blushed side of apple fruit is firmer and has a higher sugar and nitrogen content but less moisture than the green side.

Willaman (1926) correlated skin texture and flesh texture with the degree to which the fruit is infected ; a tough skin gives a " high puncture value " and a firm flesh a " low penetration value ". Resistant varieties of plums have a higher crude fibre and pentosan content than the susceptible varieties, especially in the ripe fruit (Willaman *et al.,* 1925).

Correlation between acidity and resistance has been claimed. Wahl (1926) states that La Claimanteuse and Beauty of Boskoop apples, which are almost immune from *Monilia fructigena* have a higher acidity and lower sugar content than several susceptible varieties including White Astrachan, and Katser (1933) found that a resistant variety of apple had a higher *p*H value than less resistant varieties.

Dementyeva (1951) claims that hard pruning to induce vigorous growth in apricots increases resistance. A possible explanation of this result, however, is that much infective material would be removed by hard pruning.

MODE OF ENTRY INTO HOST PLANT

In fruit infection the brown rot fungi usually enter through some previous injury, such as those mentioned above, and some writers have stated that a wound is essential. It has been proved, however, that a definite rupture of the skin is not aways necessary. Cooley (1914) found that ripe plums could be infected by placing a spore suspension on the unpunctured skin. Valleau (1915), too, showed that *Sclerotinia fructicola* could enter plums without skin injuries, the hyphae entering through the stomata and lenticels. Curtis (1928) in New Zealand, when investigating the mode of entry (also of *S. fructicola*) through the skin of various stone fruits, found that the most usual method of *effective* entry of the germ tube was through the stomata on plums and apricots, down a hair socket on peaches, and through the cuticle on apricots, cherry and nectarines.

In America, peaches are sometimes brushed to remove the pubescence of hairs in order that the fruit will present a more attractive pack. The effect of this in relation to brown rot infection was examined by M. A. Smith (1936*a*, 1936*b*), who confirmed that on unbrushed surfaces infection by *S. fructicola* occurs mostly by way of hair sockets, that stomatal infection was uncommon, and that direct infection through the cuticle was rare. In the brushed fruit he could, as a rule, easily trace the germ tubes as they grew downward along the broken hair stubs, between the stub and the adjacent buffer cell to the point of attachment of the stub and then into the tissue beneath. He states that the rapidity with which infection occurs on the brushed fruit was apparently correlated with the relative susceptibility of the brushed fruit surface to germinating conidia of *S. fructicola.*

That *Sclerotinia fructigena* can enter apples through the uninjured skin by way of the lenticels was shown by Horne (1933), using a special technique, by means of which the conidia were kept moist long enough for the germ tubes to penetrate into the flesh.

When fruits are clustered, so that they touch one another, there is likely to be greater loss from brown rot than when they hang freely. If one fruit of a cluster becomes infected through any injury the rot can then extend by contact to all in the cluster without any further wounds. A pad of mycelium develops between an infected fruit and another touching it at the point of contact, and from this pad the hyphae are able to pass through the skin of the sound fruit, possibly by direct penetration or perhaps through stomata or lenticels ; it is likely that this penetration is aided by a film of moisture at the place of contact.

Leaf infection, too, is more easily effected through wounds than through the uninjured epidermis ; experiments by Drummond (1934) suggest that plum leaves may under certain conditions become infected through the leaf glands and the uninjured lamina.

In flowers, the stigmas appear to be the organs most susceptible to attack. In peaches, however, Roberts and Dunegan (1926) found experimentally that all parts of the flower (sepals, petals, stamens, and stigma) may be infected with conidia of *S. fructicola,* and this has been confirmed by Weaver (1950). It has been observed that infection of petals can be induced in pear flowers by *S. laxa* (Wormald, 1930*b*) and direct infection of unopened flowers of Morello cherry (Wormald, 1931) and of apricot (Joëssel, 1932*a*).

Shoots are invaded by way of the leaves to cause wither tip and spur blight. Inoculations through punctures on the lower leaves of shoots of plum rootstock varieties grown as stools, with the shoots then earthed-up to cover the inoculated leaves, produced infection resulting in lesions of the shoots themselves (p. 72).

Branches become infected from shoots and particularly from flowering spurs. *S. fructigena* enters spurs and branches by way of the fruit.

The cankers that arise on branches are of limited development. In apple, plum and peach it has been noticed that infection proceeds rapidly for a time, but after a few weeks ceases and, if the canker has not girdled the branch by that time, callus develops round its periphery ; often a brown rot canker shows well developed callus at the end of the season in which infection occurred.

In America, cankers on peach trees, following infection of the flowers by *S. fructicola,* have been studied and it has been found that although the development of the brown rot cankers is of limited duration they serve as infection courts for the more destructive perennial cankers, caused by *Valsa* spp. (McCubbin, 1918 ; Willison & Chamberlain, 1935).

CULTURE, PHYSIOLOGY AND BIOCHEMISTRY OF THE BROWN ROT FUNGI

The brown rot fungi are favourite subjects for cultural and physiological studies. They grow well in pure cultures on media prepared from plant decoctions and on synthetic media and they often show interesting reactions to the various conditions to which they may be subjected.

Culture

As with other fungi used in cultural studies it is necessary to work with isolates that are known to be " pure ". To ensure purity strains each must be derived from a single spore or conidium. The isolation of conidia of the brown rot fungi is comparatively easy, for individual conidia can be seen without difficulty under a fairly low power of the microscope.

In a method recommended by Dunn (1924), conidia are transferred with a platinum loop to drops of water on slides. These suspensions are then diluted by transfers to other drops from which the transference of single spores is made with a " microloop " prepared from glass rod or tubing drawn out and bent to form a minute flat loop approximately 1·1 mm. long and 0·5 mm. wide at the widest part (inside measurement). A drop in such a loop can be examined under a microscope and those drops with single spores are selected for placing on agar slants.

A method used, with satisfactory results by the present writer, was described in 1919. Briefly it is as follows. Spores from a suspension of conidia in a watchglass are transferred in small drops of water with a platinum loop to open agar plates and examined quickly under the microscope before the drops evaporate. Each drop which appears to show a single spore is marked with a ring of ink on the bottom of the dish. The lids are then replaced and the plates kept at room temperature until the following day when the ringed spots are examined microscopically from below. Spores found to be isolated and germinating can then be readily determined and on the second or third day, when isolation is confirmed, the " sporelings " are transferred to other plates or to culture tubes.

The problem of keeping cultures viable for reasonably long periods so that transfers of isolated strains need not be made at frequent intervals is one of practical significance for the research worker who is comparing the behaviour of a large number of strains. In this connection Wernham and Miller (1948) found that cultures of *Sclerotinia fructicola* could be kept satisfactorily for two years under mineral oil without transfer, but no test was made for pathogenicity.

Physiology

The physiology of the brown rot fungi, with reference to the relation between the parasites and their host plants, was reviewed by Zimmerman (1927), but various aspects of their cultural relations, their reaction to the environment, and their parasitism have come under research since that time. Some aspects of their physiology are summarized by Hawker (1950) particularly with reference to observations on sporulation and zoning.

These fungi grow well in pure culture on prune agar, potato dextrose agar, Czapek's agar, Brown's medium, and steamed potato plugs. On such media *Sclerotinia fructicola* produces conidia readily, *S. laxa* and *S. fructigena* less freely ; thus on prune agar *S. laxa* and *S. fructigena* produce no conidia or very few, while *S. fructicola,* under similar conditions, produces conidia abundantly. Steamed potato is a good medium for conidia production, and Wiltshire (1920) has shown that potato soaked in 7·5 per cent malic acid is a better medium than plain potato for *S. fructigena.*

On culture plates of prune agar or potato dextrose agar *S. laxa* tends to grow in concentric zones. When the culture has reached a diameter of 2–3 cm. growth is checked, but is soon resumed as fan-shaped lobes grow out ; these coalesce to form another zone, and the process may be repeated several times. *S. fructicola* under similar conditions grows more rapidly and uniformly.

The concentric rings of conidial fructifications shown by *S. fructigena* on infected fruit (especially the pome fruits, apple, pear and quince) are produced also, under certain conditions, in culture, and the response of the fungus to external conditions resulting in their formation has been investigated. Molz (1907) showed that light was necessary for the development of conidia in *S. fructigena* and that the concentric rings of conidial fructifications are a result of the daily alternation of light and darkness. This has been confirmed more recently by Hall (1933) who found that conidia production in *S. laxa* also was favoured by light. Dickson (1939) showed that *S. fructigena,* when grown at constant temperature in various equal alternating periods, produced some zonation with 12 hourly alternations but at shorter periods the growth was irregular ; growth was slowest in total darkness.

Low temperatures approaching freezing point retard but do not entirely stop growth. The optimum temperature for growth is about 25°C., thus at ordinary summer temperatures, rot of infected fruit advances rapidly, while cold storage does not altogether inhibit it. Brooks and Cooley (1917) found that "*S. cinerea*" produced measurable rots at 5°C. in one week and at 0°C. in two weeks, and later (1921) found that a temperature of 10°C. held *Monilia* in check for 1–2 days, 7½°C. for 3 days, 5°C. for 4 days and 2½°C. for 6 days.

Ames (1915) investigated the cardinal points of temperatures for "*Monilia fructigena*" [?*Sclerotinia fructicola*] and found that the conidia germinated at 0°C., but that growth was slow ; that the optimum temperature for germination was 25°C., and the thermal death point 51–53°C. Brooks (1906) had noted that the action of toxic agents increased rapidly with the rise in temperature. Fish (1930) found that *S. americana* took 17 days after infection to develop into a spot 1 cm. in diameter at 32°F. and only 1 day at 71°F. According to Ezekiel (1924*b*) the minimum, optimum, and maximum temperatures for growth in culture of *S. fructigena, S. laxa*, and *S. fructicola* fall near 3°C., 25°C., and 33°C., respectively.

Bucksteeg (1940) inoculated apples, plums and cherries with *S. fructigena* and *S. laxa* and held them at varying low temperatures. The conidia were found to be viable and capable of inducing infection after 6 months' exposure to a temperature of − 14 to − 18°C., though their virulence, as judged by the rapidity of the attack on inoculated apples, gradually declined under these conditions. No viable conidia were found after 10 weeks on diseased fruits overwintering in the open.

Schneider-Orelli (1912) working with *S. fructigena*, found that in plate cultures growth was slow at 0°C., and rapid at 9·5–18°C. On inoculated apples there was rapid growth at 14°C., and it was fairly rapid at 4·5°C. Porter (1935), working with *S. fructicola* and other fungi, found that irradiation by phosphorescent light from zinc sulphate or luminous paint, either from the inside or outside of the culture dishes, generally caused a temporary inhibition of growth and increased sporulation. Thanos (1951) showed that germination was significantly reduced when a spore suspension in nutrient broth was exposed for 5 minutes to ultra-violet light, and a longer exposure killed the spores.

Clayton (1942), who studied the effect of controlled humidity, found that the conidia of *S. fructicola* on a non-nutritive surface apparently required contact with water before they were able to germinate. They germinated well in re-distilled water but failed to germinate in tests for 72 hours on glass, paraffin, or quartz at relative humidities of 100 per cent or below.

The pectin relations of the brown rot fungi, particularly with reference to the pectin enzymes, have been studied. Much of this work has been carried out by Willaman and his collaborators using *S. fructicola* (usually under the name "*S. cinerea*"). Willaman (1920*b*) found that when it is grown on a fruit juice containing soluble pectin it coagulates the pectin to a gel of calcium pectate by means of the enzyme pectase; when the fungus invades a tissue it follows the line of the middle lamella by dissolving out the latter with the enzyme pectosinase. Davison and Willaman (1927) prepared pectase and pectinase from the mycelium, but were unable to detect the presence of protopectinase in any of their strains of "*S. cinerea*", though Muhleman (1925) claimed to have demonstrated that a pectinase was present that appears to have been protopectinase (pectosinase), as defined by Davison and Willaman. A similar pectinase (or pectosinase) has been found in *S. fructigena* by Menon (1934). Jirak (1942) states that methoxylase (pectase) is the principal constituent of the enzyme complex of *S. cinerea* and *S. fructigena*, and Lin (1948*b*) that *S. fructicola* secretes large amounts of protopectinase to dissolve the middle lamella, but little or no toxic substance to kill the plant cell.

Teller and Mader (1947) grew three isolates of *Sclerotinia* spp. which were weakly, moderately and strongly virulent as judged by the rates at which they rotted apples. The *p*H of the medium was not changed by the isolates, remaining at 3·4–3·6, but the total acidity was increased.

Tests for the secretion of oxidase gave little or no reaction with strains of *Sclerotinia laxa* from plum and cherry but a pronounced reaction was given by strains of *S. laxa* f. *mali* and by *S. fructicola* (Wormald, 1920*a*; Ezekiel, 1924*b*).

Vitamins

From certain cultural studies Willaman (1920*a*) concluded that a substance of the nature of a vitamin is necessary for growth in "*S. cinerea*", and Gortner (1920) that the juice of fruits contains two vitamins necessary for the development of the fungus, one required for vegetative growth the other for reproduction.

Kögl and Fries (1937) found that *S. cinerea* was one of a number of fungi of which the growth was stimulated by the addition to a synthetic culture medium of one or more of the growth substances biotin, meso-inosital and aneurin. *Monilinia fructicola* is heterotropic for certain vitamins (Barnett & Lilly, 1947).

Serology

Link and Wilcox (1933) applied serological tests in the differentiation of certain discomycetes. *Sclerotinia fructicola* and *S. sclerotiorum,* and two strains of *S. fructicola* were differentiated from each other by the precipitin absorption test.

Many other physiological and biochemical aspects of the brown rot fungi themselves and their mode of parasitism have come under investigation, e.g.: the chemical changes in fruit induced by infection (Hawkins, 1915 ; Chrzaszy & Schillak, 1936) ; effect of CO_2 on growth (Brown, 1922) ; effect of acids on growth (Dunn, 1926) ; inhibitory effect of increased concentration of oxygen (Karsner & Saphir, 1926) ; interaction of the brown rot fungi with other fungi (Machacek, 1928: Vasudeva, 1930*b*: D'Aeth, 1937) ; respiration of conidia during germination (Frampton, 1941) ; the differentiation of cultural types by means of the hydrogen-ion concentration in cultures (Mader & Teller, 1947) ; effect of modified air (Lin, 1948*a*) ; increase in temperature and in CO_2 production in infected fruit (Fischer, 1950*a,* 1950*b*).

Other physiological aspects of the brown rot fungi are in relation to their reactions to toxic substances, particularly those that have some bearing on the control of the diseases, and many investigations have been devoted to the behaviour of the spores in fungistatic or fungicidal solutions. Until comparatively recently most of these tests were carried out with sulphur and sulphur preparations and the simpler copper compounds, but in late years some attention has been given to the toxicity of antibiotic substances, and the new synthetic organic compounds have received intensive study.

ANTAGONISM AND ANTIBIOTIC EFFECTS IN RELATION TO THE BROWN ROT FUNGI

Recent studies in the physiology of fungi include investigations on antagonism, and on the antibiotic action of the products of certain plant species. Since the discovery of the remarkable results obtained with penicillin in its therapeutic application to certain infectious diseases in man, the antibiotic nature of the products of organisms other than *Penicillium* have been examined, some of them in relation to plant pathogens, including the brown rot fungi. The toxicity of some of these antibiotics has suggested that they might be used as fungicides ; up to the present there has been little success in this direction, though one of them, acti-dione, in a preliminary field trial did show some control of brown rot (see below).

A recent review on antibiotics with special reference to their effect on bacterial and fungal plant pathogens has been compiled by Köhler (1950), with brief mention of the action of acti-dione and of subtilin on *Sclerotinia fructicola.*

A substance antagonistic to a number of bacteria and fungi, including *Sclerotinia fructicola,* was detected by Lucas *et al.* (1946) in extracts from cabbage seeds. An extract from *Impatiens balsamina* containing 2-methoxy-1,4-naphthoquinone has also proved antibiotic to *Sclerotinia fructicola* and other pathogenic organisms (Little *et al.,* 1948), and it was suggested that this compound, the synthesis of which is relatively simple, would have value as a fungicide ; it was also found (Little *et al.,* 1949) that the substitution of a sulphur atom for oxygen in the molecule resulted in a marked increase in anti-fungal activity.

Juglone (5-hydroxy-1,4-naphthoquinone), the oxydized form of hydrojuglone found in *Juglans* spp., is not only toxic to higher plants growing near walnut trees but also to certain fungi. In laboratory trials with *S. fructicola* as one of the test organisms its toxicity approached that of copper in Bordeaux mixture (Gries, 1943).

Vasudeva (1930*a*) considered that the failure of the spores of *Monilia fructigena* to germinate when placed in wounds on onion was due to the presence of a thermolabile substance which can be extracted with chloroform or ether. This suggests the presence in onion scales of a substance antibiotic to *M. fructigena.*

In a study of the mutual antagonism of species of bacteria and fungi, Darpoux and Faivre-Amiot (1949*a*) included *Sclerotinia fructigena* and *S. cinerea* among those which are particularly sensitive to the antibiotic activity of a number of other organisms. They also found (1949*b*) that an actinomycete markedly inhibited the growth of various fungi in cultures including strains of *S. cinerea* from plum and cherry. Antagonism of *Botrytis allii* towards *Monilia fructigena* was demonstrated by Vasudeva (1930*b*) who found that, when a mixture of spores of the two fungi was used as inoculum on apples, the vigour of parasitic attack by the brown rot organism was markedly reduced.

A crystalline toxin, gliotoxin, derived from *Gliocladium fimbriatum,* is antibiotic to germinating spores of *Sclerotinia fructicola,* its fungicidal effect being greater than $CuSO_4$ but less than that of $HgCl_2$ (Weindling, 1941).

In tests on the conidia of *S. fructicola* with heavy metal salts of kojic acid (an antibiotic derived from *Aspergillus flavus* and other fungi) the cadmium salt was comparable in activity with Bordeaux mixture, the salts of Cu, Zn, Sn, Hg, Fe, Co, Ni, and Mn being less active (O'Kane, 1950).

A substance, referred to as endomycin, isolated from *Streptomyces* sp. was found by Gottlieb *et al.* (1951) to inhibit growth of *Sclerotinia fructicola* on streaked plates and in shake-culture fluids.

Leben and Keitt (1947, 1948) found that a species of *Streptomyces* was antagonistic on agar to 33 fungi including *Sclerotinia fructicola,* the inhibition point for one isolation being 1·6μgrm. per ml., and 0·4 for another. The antibiotic substance was tentatively named " antimycin ".

Cycloheximide, an antibiotic obtained from cultures in which streptomycin-producing strains of *Streptomyces griseus* are cultivated, and prepared under the trade name Acti-dione, has received some attention because of its fungicidal properties. Vaughn and his co-workers (1949) concluded from laboratory assays and preliminary tests that it was worthy of extensive trials. The growth of *Sclerotinia fructicola* was strongly inhibited by concentrations of 100 p.p.m. in potato dextrose agar, and in dipping experiments for post-harvest control of brown rot of peaches; fruit dipped in 20, 50, or 100 p.p.m. solution for 3, 5, or 10 min. developed 10 per cent rot while untreated peaches developed 90 per cent. In Blakeslee's medium the growth of *S. fructicola* was retarded at concentrations between 1 and 10 p.p.m. and was inhibited at 1000 p.p.m. (Gottlieb *et al.*, 1950). Used in field trials at 20 p.p.m. it showed indications of some control of brown rot, but in a second test it was inferior to wettable sulphur 5 lb. in 100 gal. It would appear from the severe injury to nearly ripe fruit (it caused cracking of peaches) that Acti-dione is of doubtful value as a peach brown rot spray (Peterson & Cation, 1950).

Musarin, a toxic substance produced in culture fluids by "Meredith's actinomycete" is said to be one of the most potent antifungal antibiotics, though as an antibacterial agent it is less effective (Arnstein *et al.*, 1948). It induced complete inhibition of conidia of *Sclerotina fructigena* at a dilution of 1/800,000. Streptothricin was also active against *S. fructigena* and other fungi, but streptomycin was found to be inactive.

Certain bacteria appear to be antibiotic to the brown rot fungi. Michener and Snell (1949) find that *Bacillus subtilis* cultures secrete two substances antibiotic to a number of fungi, including *Sclerotinia fructicola,* and Keil (1950) reports that two bacteria, *Sporobacterium fungostaticum* and *Bacillus antimycoides,* isolated from the air at Mainz, strongly inhibited growth of *Monilia cinerea* (and other fungi) on molasses agar cultures. The inhibitory power of tyrothricin, an antibiotic derived from *Bacillus brevis,* was investigated by Ciferri and Scaramuzzi (1949) on the germination of conidia of four species of fungi; the dosage response curves were regular and almost parallel for *Botrytis cinerea* and *Monilia fructigena*: LD50 about 1·3μgrm. per 100 ml.

Warren *et al.* (1951) isolated an actinomycete from soil in which tomato plants had been grown and sprayed with 2, 4–D; it was found to be antibiotic towards a number of plant pathogens, including *Sclerotinia cinerea.*

LABORATORY METHODS—TOXICITY TESTS

In order to obtain information on the toxicity of known or potential fungicides, laboratory methods with toxic substances have been elaborated for plate and tube cultures and, more especially, for spore germination tests. The brown rot fungi have received much attention in this connection for they are important economically as causing great losses in the fruit growing industry and so merit examination in the direction of improving control measures, and, for laboratory tests, they are easily grown on prepared culture media and readily provide a supply of spores as required. Other advantages are the reasonably large size of the spores which facilitates microscopic examination, their sensitiveness to certain chemicals and their ready germination in water.

Although most of these laboratory tests with the brown rot fungi have been undertaken in the United States, *Sclerotinia fructicola* being one of the tests organisms, some work has been carried out in Europe on *S. fructigena* and *S. laxa.*

Work started at the Cornell University and continued at the Boyce Thompson Institute by McCallan and his colleagues, who have made great use of *S. fructicola,* is summarized by McCallan (see Crocker, 1948). Other papers published in that series since 1948, also mentioning *S. fructicola* as one of the test organisms, are by McNew and Burchfield (1950, 1951), Schmitt (1951), Goldsworthy *et al.* (1933–1949) and Horsfall and Rich (1951). The more interesting of these contributions relating to the brown rot problem are listed in the references to this bulletin, the titles usually indicating their scope ; some of them will be mentioned below.

The slide germination method as developed at the Boyce Thompson Institute with particular reference to *Sclerotinia fructicola* has been accepted as a standard by the American Phytopathological Society (1943*a*). The method is designed to evaluate the fungistatic properties of protective fungicides by placing spores of certain fungi on glass slides in water in the presence of the chemical to be tested. All conditions are rigidly controlled and, except for the chemical, are favourable for spore germination. The chemical may be applied either by the test-tube dilution technique or by the horizontal sprayer or settling-tower technique (McCallan & Wilcoxon, 1940*b*). The results are expressed as concentration of chemical necessary to inhibit the germination of 50 per cent of the spores, i.e. the LD50, or as the LD95 ; the former is more precise.

This method of evaluation has been adopted by a number of workers in the United States and in Europe, one or other of the brown rot fungi frequently being employed.

McCallan and Wilcoxon (1933) introduced the concept of the " toxicity surface ", which can be shown by a model in three dimensions to show the relation between the three most important variables, time, concentration and percentage response. They show the form of the " toxicity surface " for the germination of conidia of *S. fructicola* in the presence of copper sulphate as an example of a soluble toxic agent and sulphur dust as an example of an insoluble one.

With reference to temperature as it affects toxicity in laboratory tests, Brooks (1906), using sulphuric acid, nitric acid and copper sulphate in sugar beet decoction, and McClennan (1942), using copper sulphate and particulate suphur, found that the substances tested were least effective at the optimum temperature for the germination of spores of the test organism (21° C. for *S. fructicola*).

Before reviewing those substances that have proved to be toxic to the brown rot fungi some mention should first be made of the germination of the conidia under non-toxic conditions, as investigated by a number of workers, whose results may be summarized as follows:

The conidia of *S. fructicola* will not germinate in moist air ; they require precipitated moisture. In a drop of distilled water there was 100 per cent germination ; on dry slides, in a moist chamber, none germinated. The time necessary for the germination of the conidia was 5 hours under optimum conditions. They germinate quite as well in sunlight as in darkness if conditions of temperature and moisture are near the optimum (Doran, 1922). Yarwood (1936) confirmed that the conidia failed to germinate on dry slides even in an atmosphere of high relative humidity.

The conidia usually do not germinate in pure water ; the addition of a small amount of carbohydrate (e.g. dextrose) or ethyl alcohol, however, brings about vigorous germination. Conidia failed to germinate satisfactorily in distilled water. Of several nutrients tested, potato-dextrose-agar extract proved to be most highly stimulatory. A method is described of applying conidia to glass circles attached to glass slides with petroleum (Peterson, 1941).

A linear relationship of increasing concentration of orange juice, citric acid and sodium and potassium salts, and bean extract to increasing LD50 values of copper, nickel, and zinc compounds was found by Miller (1950).

With regard to *p*H limits and temperature, the widest reaction range—1·4–7·2—occurred at 21°C. The optimum *p*H for *S. fructicola* on potato dextrose agar at 10–24°C., 25–26°C. and 27–30°C. were near 3·79, 3·79–4·6, and near 5·8 respectively (Tilford, 1936).

Gerhardt *et al.* (1945) found that temperatures of 40°F. and 32°F. exerted a marked inhibitory effect on the germination, the conidia of *S. fructicola* requiring from 12 to 13 hours for germination at 40°F.

In Europe, Leszczenko (1928) remarked that, with *Sclerotinia fructigena* (among other fungi), germination of conidia was more active and more rapid in rain, tap, or ordinary distilled water than in twice-distilled water. This effect is attributed to the slightly acid reaction of the rain and distilled water, and to the slightly alkaline reaction of the tap water, the retardation in twice-distilled water being probably due to neutral reaction.

Copper-containing Substances

In the early studies of the reaction of the brown rot fungi to fungicides the simpler copper compounds were used because of the extensive use of copper sulphate in the preparation of Bordeaux and Burgundy mixtures for the control of plant diseases caused by fungi, including the brown rot organisms. Thus, in 1889, Galloway showed one-fifth per cent of copper sulphate was sufficient to prevent the germination of the conidia of "*Monilia fructigena*" [?*Sclerotinia fructicola*]. More recently Faes and Staehelin (1925*a*, 1925*b*) tested the effect of a number of metallic salts on *Monilia fructigena* spores and found that copper sulphate and particularly nickel sulphate were more toxic than the zinc or iron sulphates.

Laboratory tests at the Cornell University showed the toxicity of copper sulphate solutions to the conidia of *Sclerotinia fructicola* to be directly correlated with the total content of copper (calculated on the basis of concentration and volume of the solution), and inversely related to the number of spores. Preliminary observations suggested that the excretions of the germinating spores were very slightly acid, to the extent of about the equivalent of N/20,000 HCl (McCallan, 1930).

McCallan (1948) determined the copper sulphate toxicity curve from extensive data obtained for the dosage response of copper sulphate on inhibition of spore germination of *S. fructicola*. Germ tube length, expressed as percentage of control when plotted against percentage germination, gives a sigmoid curve with germ tube length increasing with percentage germination.

That copper sulphate itself is very toxic in low concentration was shown by Goldsworthy and Green (1938). Conidia of *S. fructicola* were injured at concentrations of ionic copper as low as 0·25 p.p.m. administered in a dynamic system (continuous supply); in the static system toxicity varied according to the power of the various media to fix and inactivate copper ions. In 1933 they had found that finely ground copper oxide, copper silicate, and copper phosphate were toxic to conidia of *S. fructicola*.

On malt agar plates certain copper-lime-arsenic mixtures were highly toxic to *S. fructicola* (and other fungi), and the toxicity could be varied through a wide range by modifications of the formulae (Palmiter & Keitt, 1937).

Whaley and Harry (1951), in a study of chromates using the spore-inhibition technique of the American Phytopathological Society (and so probably including *S. fructicola* as one of the test organisms although it is not mentioned by name), list 21 copper chromate complexes with their LD50 values.

Blumer and Kundert (1950) devised an elaborate apparatus for determining the LD50 value for copper preparations. *Monilia fructigena* can be used for these tests at an age of 7–10 days, but, with the strains used, germination in distilled water was very irregular and varied from 70–90 per cent. The copper-sensitiveness of *Monilia* showed an interesting development during the course of the tests. At first this fungus, in comparison with others tested, was very insensitive towards copper, but the LD50 value sank on repetition, indicating a considerable increase in its copper-sensitiveness.

The available copper in Bordeaux mixture residues subjected to weathering, amounting to at least 4 p.p.m. by volume of the original mixture, was lethal to germinating and germinated spores of *Sclerotinia fructicola* (Goldsworthy & Green, 1936). When the quantity of copper in the residue is small, it may be exhausted by the conidia germinating first, the late ones escaping injury (Goldsworthy & Green, 1936). When the residues were weathered for 2 weeks the compounds were lethal to conidia of *S. fructicola* generally in accordance with the initial ionic copper content of the residues (Goldsworthy, 1944).

Wilson (1939) tested spores of *S. fructicola* on dried films made from successively diluted Bordeaux mixture and oil-Bordeaux; at the greater dilutions oil-Bordeaux appeared to be somewhat less toxic than Bordeaux mixture itself.

It is interesting to note that McCallan and Wilcoxon (1938) concluded, from a review of literature and from their own results with copper compounds, that none is equal to Bordeaux mixture in fungicidal action.

Copper-containing compounds more complex than those already mentioned have also been tested for their toxicity to the brown rot fungi, particularly *S. fructicola*. In tests with 8-quinolinol and its copper derivative both were found to be highly toxic to *S. fructicola* with an LD50 range of less than 1·5 μgrm. per sq. cm. of glass-like surface (Powell, 1946).

Copper resinate in " Palustrex " (a pine-tar oil solvent) in germination tests with "*Monilia*" [?*Sclerotinia fructicola*] compared favourably with copper sulphate (de Ong, 1932).

In tests with copper naphthenate Youganova (1946) found it to have pronounced toxicity to conidia of "*Monilia*" [*Sclerotinia laxa*]. On glass slides at 0·3, 0·6 and 0·8 per cent in an oil-kaolin emulsion it was about as toxic as 1 per cent Bordeaux mixture. She reported promising results with this preparation in spraying trials against blossom wilt of apricots.

Zinc-oxyquinolene and cupro-oxyquinolene complex compounds were found by Ciferri and Scaramuzzi (1947) to have a marked fungicidal effect on conidial germination of "*Sclerotinia fructicola*" (!).

In laboratory perfusion experiments by Goldsworthy *et al.* (1942) all the copper xanthates tested proved toxic to some extent to conidia of *S. fructicola*, the ethyl and butyl xanthates being the most toxic. The toxicity was of the same degree as that exhibited by a solution containing about 0·50 p.p.m. of copper. Spray residues containing the ethyl and isoamyl xanthates were toxic to the conidia.

Another line of investigations is one relating to the counter-action of certain substances to the toxicity of copper sulphate towards the spores of brown rot fungi.

P. B. Marsh (1945) found that the conidia of *S. fructicola* have a very high affinity for copper, being able to concentrate it within themselves from a very dilute solution, and that calcium chloride, magnesium sulphate or potassium chloride may counteract the inhibitory effect of copper sulphate on the germination of the spores.

Again, the toxic effect of $CuSO_4$ solutions on respiration and germination of the conidia is partially or completely reversed by the addition of solutions or sulphydryl-containing compounds. The completion of the reversal is dependent on the molar ratio or antidote to $CuSO_4$, the length of time elapsing between the addition of Cu solution and the addition of the antidote, and the type of antidote.

An example of synergism is recorded by Ciferri (1950) for copper sulphate and ammonium hexanitrodiphenylamine ; he found that their toxicity was increased when the two were used together on six fungi tested, including "*Sclerotinia fructicola*" but not on *Plasmopara viticola*.

Lin (1940) showed that electrolytes antidote the toxicity of copper and promote spore germination.

Sulphur and Sulphur Compounds

Sulphur, in various forms and combinations, has often been used in laboratory tests because of its importance in horticultural practice for disease control, and also because its marked toxic action towards fungi, particularly the Powdery Mildews, has interested many research workers who have sought to explain its fungicidal action. Some of these studies have been summarized by Horsfall (1945) or are mentioned in recent papers on the subject (e.g. Baldwin, 1950).

Only comparatively recently has the action of sulphur on the brown rot fungi been investigated under controlled laboratory conditions. Barker *et al.*, (1920) mentioned *Sclerotinia fructigena* as one of the organisms used in an investigation of the action of sulphur and some of its compounds. The conidia were found to be very sensitive (100 per cent inhibition) when placed in hanging drops of water containing particles of sulphur. Germination was inhibited also by low concentrations of sulphuretted hydrogen, sulphur dioxide and sulphuric acid. In examining the sphere of influence of sulphur particles on culture plates it was seen that with *S. fructigena* there were well-defined zones round the masses of sulphur within which no germination took place. Later Barker (1928) demonstrated toxicity to conidia of *S. fructigena* of a gaseous sulphur product, probably sulphuretted hydrogen, and R. W. Marsh (1929) found that H_2S inhibited the germination of *Monilia fructigena* spores at a concentration of 1 in 3,000 and those of *Monilia cinerea* at 1 in 40,000.

Many papers on the fungicidal action of sulphur and its compounds using *Sclerotinia fructicola* as one of the test organisms have been published in the United States. Young (1922, 1923) studied the toxic properties of sulphur on several fungi including *S. cinerea* [*S. fructicola*]. Flowers of sulphur were found to be directly toxic to this fungus ; finely-ground flowers of sulphur were more toxic than unground sulphur, the range of greatest toxicity being *p*H 4·2 to 5·4. The hydrophilic form of colloidal sulphur proved to be highly toxic to *S. fructicola*, while the hydrophobic solution exerted only approximately the same slight toxic action as ground sulphur. It was considered that the hydrophilic colloidal sulphur contains the toxic substance produced by the ordinary forms of sulphur ; it was toxic in van Tieghem's cells when separated 8 mm. from the spores.

A series of articles was issued from the Boyce Thompson Institute (1930–1932) on the toxicity of sulphur dusts and of pentathionic acid. This acid was not considered an important factor in the fungicidal action of sulphur; its neutral salts are not toxic to conidia of *S. fructicola* except when treated with sodium hydroxide which destroys the pentathionic and forms colloidal sulphur (Wilcoxon & McCallan, 1930). McCallan and Wilcoxon (1931) explain the fungicidal action of sulphur by suggesting that sulphur vapour diffuses from the sulphur particles to the spores and is reduced within the spore to form hydrogen sulphide. The toxicity of pentathionic acid was investigated by Young and his collaborators who considered it to be the toxic factor of sulphur, the natural oxidation and dissolving of sulphur giving a continuous yield (Young & Williams, 1928). In testing various sulphur acids for toxicity to *S. fructicola*, Liming and Young (1930) found that pentathionic acid was the only one showing a marked fungicidal action. Sulphur freed from pentathionic acid, either by strong alkali or acid or by washing, was not toxic. Acid-free sulphur aerated for 8 hours regained its normal toxicity, indicating that this property is a result of the oxidation of sulphur. Liming (1932, 1933) found that pentathionic acid is associated with sulphur and is toxic.

That the toxicity of sulphur dusts increases with the fineness of subdivision has been shown by a number of workers—e.g. Wilcoxon and McCallan (1931), Hamilton *et al.* (1943)—and this led manufacturers to produce sulphur in very fine particles for use as dusts or in such preparations as colloidal sulphur.

Various proprietary sulphur dusts and wettable sulphur preparations are listed by Hamilton (1931) with particular reference to apple scab control, but he mentions also certain studies on conidia of *S. fructicola*. The toxicity of a number of sulphur dusts to the conidia across the space in van Tieghem's cells was indicated; the greatest inhibitory action under these conditions seemed to be at a temperature of about 20°C. An amorphous form of sulphur (particles about 2μ in diameter), produced by evaporating an acetone solution of sulphur, was highly toxic to conidia of *S. fructicola*—LD50 of 0·13μg per sq. cm. (Feichtmeir, 1949).

Wallace *et al.* (1911) proved that the addition of lead arsenate increases the fungicidal value of lime sulphur, one of the fungi tested being "*Sclerotinia fructigena*" [?*fructicola*], and Hamilton (1931) found lead arsenate to increase the toxicity of Supersulfodust and lime sulphur against conidia of *S. fructicola* in hanging drops at 20°C. and 17°C.

The fungicidal activity of sulphur plus urea is greater than that of sulphur alone (Stoddart, 1950). Against *Sclerotinia* [?*fructicola*] a mixture of sulphur and urea at the LD50 level was approximately twice as effective as sulphur alone, and the tests suggest that there is evidence of synergism between urea and sulphur.

Stewart and Standen (1946) in tests with polymeric organic polysulphides used the spore germination method and also an agar plate method, *S. fructicola* being one of the fungi; they found the polysulphides to be fungicidal, toxicity increasing with the sulphur content of the polymers. The substitution of a sulphur atom for oxygen in the 2-methoxy-1,4-naphthoquinone molecule resulted in a marked increase in anti-fungal activity tested against *Monilinia fructicola* (Little *et al.*, 1949).

Other sulphur-containing substances will be mentioned later under organic compounds.

Organic Compounds

Organic compounds have been investigated intensively since the inception of their use as fungicides, the toxicity of some 2,000 organic or metallic organic compounds having been tested at the Boyce Thompson Institute alone (Crocker, 1948). In one series of tests, including *Sclerotinia fructicola* as one of the indicator fungi, the toxicity of 32 such compounds, including organic thiocyanates, thiazoles, resorcinol derivatives and other phenolic compounds was compared with that of copper sulphate and formaldehyde. The thiocyanates and the alkyl and acyl resorcinols were found to be highly toxic, some members of these series being about as fungicidal as copper sulphate (Wilcoxon & McCallan, 1935).

In a study of phenothiazine and its derivatives, Goldsworthy and Green (1939) found that phenothiazone was the most effective of this group, being toxic at several p.p.m. to the conidia of *S. fructicola,* and Goldsworthy *et al.* (1942) proved that 2,4-diaminophenylamine and other substituted diphenylamines were comparable in toxicity to Bordeaux mixture.

In 1949 Goldsworthy and Gertler published an account of tests on the conidia of *Monilinia fructicola* and *Glomerella cingulata* with 506 synthetic organic compounds ; of these 456 failed to show any toxic effect and 47 were toxic to conidia of both fungi in some degree. The toxicity, however, of most of these compounds was so slight that they were of no real value as orchard fungicides, and many were so toxic to peach foliage as to be unsuitable. The ten organic compounds which appeared to be most promising as fungicides were: *p*-chloro-alpha-isonitroso-acetanilide, *p,p'*-thiodianiline, benzamide, benzimidazole, *p*-chloro-benzoic acid, 3,4-dichloro-benzoic acid, N-(*O*-methoxyphenyl)-glycinonitrite, 2,2'-methylenebis[4-chloro-phenol], 2, 2'-(2, 2, 2-trichloroethylidene)bis[4-chloro-phenol], and 4, 4'-*iso*-propylidenediphenol.

These appeared to have satisfactory fungicidal properties, are apparently stable when mixed with insecticides, lime and adjuvants, and appear safe to use on tender peach foliage. More recently (1951) they tested 412 additional synthetic organic compounds using the same two fungi ; only 5 appeared promising enough for orchard tests.

Horsfall (1951) tested 1,000 organic compounds ; toxicity to *Sclerotinia fructicola* was shown by basic $-NH_2$ compounds, by hydrazines which can react with sugars, and by phenolic hydroxyls, the $C=O$ group being especially toxic to *Sclerotinia.*

Palmiter and Hamilton (1942) published on account of a new fungicide, ferric dimethyldithiocarbamate (sold under the name of Fermate), for the control of brown rot of sweet cherries. Laboratory experiments indicated that it was more than twice as toxic as sulphur to the fungus spores. In 1943 they stated that Fermate and Jap Beetle Spray (tetramethylthiuram-disulphide) when sprayed on cellulose-nitrate-coated glass slides were equally toxic to the spores of *Sclerotinia fructicola.* Both showed two to three times greater toxicity than micronized sulphur or red copper oxide which have been used for controlling brown rot on sweet cherries. Because of these favourable initial results with Fermate, this and other dithiocarbamates have received the attention by other investigators (see particularly Feekes, 1949).

Cohen (1951) tested three phenyl mercury proprietary preparations against several organisms, including *Sclerotinia* [?*fructicola*] in spore germination tests, and found that solutions adjusted to contain equivalent amounts of the phenyl mercury group for each compound gave significantly similar results.

Horsfall and Rich (1951) assayed 165 heterocyclic nitrogen compounds by the spore-germination method against *S. fructicola*, McNew and Burchfield (1951) tested the activity of quinones, and Foote *et al.* (1949) tested a number of quinones including 8-napthaquinone.

McNew and Sundholm (1949) tested 4-nitropyrazoles and found that the more active members of the series prevented spore germination on glass slides at concentrations as low as 0·1 p.p.m., and McNew and Burchfield (1950) in testing the relationship of particle size of dichloro-naphthoquinone to its fungicidal action, stated that there was some evidence that particles $< 1\mu$ in radius were less effective on glass slides than those of 1–3μ or 3–5μ.

Of 20 mercaptothiazoles, Schmitt (1951) found that with 5 representative species of fungi (including *S. fructicola*) grown on agar plates, the benzoyl, n-butyl and acetonyl derivatives were the must fungistatic.

In a study of eradicant fungicides Carpenter (1942) used bottle cultures with a malt extract medium ; a commercial preparation of ethyl mercury phosphate (Lignason) was the most toxic of the preparations tried.

Some of these complex organic substances have been tested in Europe on *Sclerotinia fructigena* and *S. laxa*. Results of preliminary spore germination tests carried out by Byrde (1949) showed the high fungistatic action of phenyl mercury chloride, 8-hydroxyquinoline sulphate, 2:3-dichloro-1:4-naphthoquinone and 2-heptadecyl glyoxalidine on *S. laxa*. Of these compounds phenyl mercury chloride was used in field experiments. In other tests (1950) with seven fungicides the provisional mean ED-50 values (in $\mu g/cm^2$) against *S. fructigena* was as follows: Phygon "XL" 0·03 ; 8 hydroxyquinoline sulphate 0·058 ; copper 8-quinolinolate 0·074 ; Bordeaux mixture (as Cu) 0·21 ; "Experimental Fungicide 341-c" 0·33 ; lime sulphur 1·05. Continuing these tests Byrde (1952) confirmed the high toxicity of phenyl mercury chloride and of 2:3-dichloro-1:4-naphthaquinone towards conidia of *S. laxa* and *S. fructigena* and also found that several newly introduced fungicides showed promise, notably N-trichloromethylthiotetrahydrophthalimide, bis (2-hydroxy-5-chlorophenyl) sulphide and two substituted 4-nitroso-pyrazoles.

In Holland, Feekes (1949) carried out spore germination tests on 10 different fungi, including *S. fructigena* with tetramethylthiuramdisulphide, ferric dimethyldithiocarbamate, zinc dimethyldithiocarbamate with copper sulphate and mercuric chloride for comparison. The substances were compared at strengths 0·001 and 0·0001 per cent in distilled water. The fungicidal action of the dithiocarbamates was less than that of $HgCl_2$ but greater than that of $CuSO_4$.

In Italy, Ciferri and Scaramuzzi (1949) investigated the inhibitory power of tyrothricin on the germination of conidia of four species of fungi and found that the dosage response curves were most regular and almost parallel for *Monilia fructigena* and *Botrytis cinerea*: LD50 about 1·3μg per 100 ml.

Miscellaneous Preparations

A number of other substances have been shown to be toxic to the brown rot fungi in the laboratory.

Schmidt (1924*b*) testing the toxicity of soft soap solution on certain fungi including a "*Monilia*", found the fungicidal action of the solution was due to a definite degeneration of the fungal cells in contact with the soap.

Schubert and Richter (1926) in Germany examined the toxicity of hydrocarbon and phenol preparations. The hydrocarbons had no more than a repressive action on germination but the high boiling phenols (230–320°C.)

had fungicidal properties of varying degree. The *dosis letalis* of the phenols boiling at 270–275°C. for *S. fructigena* was 0·033 per cent (10 min. immersion). The conidia were not killed after 40 minutes immersion in the low-boiling phenol at 0·33 per cent.

Tomkins (1930), in England, tested conidia of *Monilia cinerea* sown on dried nutrient agar on a glass slide suspended in air in a bottle containing water or a watery solution of acetaldehyde at various concentrations and kept at a constant known temperature. At all the temperatures tested, the presence of acetaldehyde delayed the appearance of germ tubes while at any given temperature the latent period of germination increased with an increase in the concentration of the acetaldehyde. Concentrations between 3 and 5 in 10,000 usually inhibited germination but when the spores were moved to atmospheres free from acetaldehyde, they began to germinate. A concentration of 5 in 10,000 inhibited the germination of *S. cinerea* at 10°C., but not at 25°C. All were killed by 1 in 100 in 3 hours at 23°C., and in 9 hours at 10°C. The higher the concentration the longer was the time required to establish a colony and in the early stages, the slower was the rate of spread.

Janke and Beran (1933) used three weeks old cultures in prune extract or on prune extract agar when the substance was not entirely soluble. *Sclerotinia cinerea* was killed in 5 minutes by formic acid, in 30 minutes by acetic acid, in 5 minutes by butyric acid and phenol, and in 10 minutes by copper salicylate. The growth of this fungus on removal from the solution was not impaired by exposure to copper sulphate for periods up to 360 minutes, while copper chloride exercised an inhibitory effect only after 180 minutes.

Watson (1943) examined the toxicity of ozone in relation to fruit infection in cold storage. A shorter exposure (1–2 hours) to ozone was required to kill the conidia of *S. fructicola* in drops than in 5 ml. in water in a 9 cm. Petri dish. No wet spores of this fungus germinated after treatment for from 2–2½ hours.

Sclerotinia fructicola and *S. laxa* are both sensitive to diphenyl vapour and their growth was suppressed for 5 days when crystals of diphenyl were introduced into inoculated plates. The vapour had a fungistatic, not a fungicidal, effect under these conditions for when the treated inoculum was transferred to untreated plates the fungi grew out normally (Heiberg & Ramsey, 1946).

FUNGICIDES IN RELATION TO THEIR USE FOR THE CONTROL OF THE BROWN ROT DISEASES

Fungicides

When it was realized that the serious losses resulting from brown rot diseases were due to fungus infection, precautions were taken to reduce such losses. Hygienic measures were advised but it was soon found that such methods could be supplemented with advantage by the application of fungicides. At first, in Europe, copper-containing preparations, particularly Bordeaux mixture, were usually recommended but often without any evidence that they were effective. As brown rot of peaches became more and more a serious problem to American fruit-growers, Bordeaux mixture was found to be not very satisfactory since it was liable to cause damage and lime sulphur too was found to be injurious. This led to the introduction of safer preparations such as self-boiled lime sulphur, dry mix lime sulphur, and then to the many forms of elemental sulphur.

E. F. Smith (1889) mentions what must have been one of the earliest occasions when sulphur dust was used for the control of brown rot. The value of sulphur preparations in brown rot control has often been shown in the results of trials and mentioned in general terms. Anderson (1925*a*) said that brown rot in properly sprayed or dusted orchards was no longer a serious factor under Illinois conditions, and Hoerner (1930) that sulphur dusting had given such satisfactory control that it had become a regular orchard practice. But recently Lewis (1950) said that brown rot in Pennsylvania remained the major disease problem on peaches, and advocated blossom spraying.

Many proprietary sulphur dusts are on the market and it is claimed that particle size has been reduced and efficiency thereby increased. A list of sulphur spray materials for the control of apple scab, including particle sizes, is given by Hamilton and Palmiter (1951).

Among forms of elemental sulphur which have given good control of brown rot when used as sprays may be mentioned wettable sulphur (Berkeley, G. H., 1928 ; Sharvelle & Burkholder, 1947), wettable bentonite sulphur (Rudolph, 1936), Sulfuron X—a wettable sulphur (Poulos, 1950), colloidal sulphur (Young, 1923), hydrophilic colloidal sulphur (Winter & Young, 1938), flotation sulphur (Rudolph, 1936), Magnetic-70 sulphur paste (Lewis, 1950 ; Poulos, 1950).

In recent years, attention has been given to the new synthetic organic fungicides. In 1942, Palmiter and Hamilton experimented with ferric dimethyldithiocarbamate (ferbam) supplied under the trade name Fermate. When used in field trials on cherries just prior to harvest, it left no visible residue and protected the fruit from decay for several days after harvest under conditions favourable for rot development. But Roberts (1946), records that applications of Fermate just before harvest for the control of brown rot of cherries and peaches left an undesirable black residue on the picked fruit. Trials with Fermate have been carried out with more or less favourable results against brown rot of stone fruits by a number of other workers (Folke, 1950 ; Yarwood, 1948 ; Wilson & Scott, 1943*a* ; Zeller *et al.* 1942).

Other synthetic preparations which have shown promising results in trials against brown rot of stone fruit trees in the United States are:

Zerlate (Zinc dimethyldithiocarbamate)—see Hamilton, 1948; Foster, 1949; Lewis, 1950.
Puratized Agricultural Spray (phenylmercurictriethanol ammonium lactate)—see Johnson, 1950; Folke, 1950.
Isothan Q15 (lauryl-*iso*-quinolinium bromide)—see Hamilton *et al.*, 1944 ; Hopperstead *et al.*, 1945.
Bioquin 1 (Copper 8-quinolinolate)—see Morgan & Powell, 1949; Poulos & Heuberger, 1949.
Phygon XL (2, 3-dichloro-1, 4-naphthaquinone)—see Powell, 1951; Dunegan & Goldsworthy, 1948; Hamilton, 1948; Foster, 1949.
Spergon (tetrachloroquinone)—see Roberts, 1946; Palmiter & Hamilton, 1943.
Tersan (tetramethylthiuramdisulphide)—see Palmiter & Hamilton, 1943.
341-C (a glyoxalidine derivative)—see Folke, 1950.
Krenite (sodium dinitro-*o*-cresylate)—see Fungicide Committee Amer. Phytopath. Soc., 1949 and
Jap Beetle Spray (a tetramethylthiuramdisulphide preparation)—see Palmiter & Hamilton, 1945.

For comparisons of the efficacy of some of these synthetic preparations, two papers may be mentioned. During the years 1942–45, Palmiter and Hamilton tested a number of organic fungicides for the control of brown rot of sweet cherries. They found (1943) that Fermate and Jap Beetle Spray were superior to Spergon and micronized sulphur for pre-harvest sprays on sweet cherries and later, in 1945, they concluded that Fermate 1–100 is markedly superior to micronized sulphur 5–100 and that Isothan Q15 has possibilities of being a satisfactory colourless spray for sweet cherries.

Folke (1950) recorded the percentage of blighted blossom spurs in trials with the following fungicides applied at pre-blossom stage: Arathane, ferbam, phygon XL, Roccal, Puratized Agricultural Spray, lime sulphur and 341-C. Both Arathane and lime sulphur caused injury to the flower petals but no effect on fruit set could be detected.

The various investigators are not unanimous however about the value of ferbam and other synthetic preparations for the control of brown rot. Smith and Clayton (1950) state that of the new fungicides which are safe as summer sprays on peach trees, none has proved better than wettable sulphur or sulphur dust when properly used. Lewis (1950) also says that the newer fungicides are not of sufficient promise to justify their cost at this time, that good results may be obtained with phygon, but it causes skin irritation, and that Zerlate applied pre-harvest is no better than sulphur.

Times of Application

Fungicides can be applied against brown rot from winter until just before the fruit is picked or even during storage, and also for transit when, as in America and Australia, the fruit has to be taken to markets of long distances from the fruit farms.

The preparations used in orchard practice or in trials vary greatly according to the stages of development of the trees, particularly whether these are dormant, in active growth, in bloom, or when ripening their fruit.

The periods during which fungicides may be applied have been investigated by many workers in the United States for the control of brown rot of stone fruit trees and are set out below. Since growers would doubtless find that to make applications at all the times mentioned would be impracticable or unprofitable, a choice has to be made, with the result that numerous programmes or spray schedules have appeared in the bulletins issued by the various research stations. The number of spray applications recommended for the control of the brown rot diseases in Britain and on the Continent are far fewer.

DORMANT SPRAYS are applied in winter or early spring before the buds begin to swell, in order to destroy or render innocuous the sporodochia developing on organs killed the previous season.

In the United States it was shown that eradicant sprays could be used with good effect during the dormant season. Wilson (1940) found that calcium arsenite applied in winter frequently reduced development of sporodochia by 95–99 per cent; in 1942, he said arsenite injured almond trees but was satisfactory for apricots and plums.

Calcium meta-arsenite was used by Wilson and Scott (1943*b*) as a dormant spray for apricots with good effect.

Calavan (1944) effectively controlled brown rot blossom and spur blight on cherries by applications shortly before bud-break of copper-lime-mono-calcium arsenite and fish oil, supplemented in early bloom by Bordeaux mixture (3:4:50). Fish (1934) in Australia found that sodium orthodinitrocresol gave promising results in preventing sporulation of *S. fructicola* on infected twigs.

DELAYED OR LATE DORMANT SPRAYS when the flowers are emerging from the buds. A late dormant copper-lime-arsenite mixture has been recommended in Wisconsin (Anon., 1935). Keitt and Palmiter (1937) obtained favourable results on plum branches bearing mummified fruit by applying eradicant sprays when the blossom buds had expanded but not separated in the clusters. Wilson and Serr (1938) sprayed apricot trees with sodium arsenite at the time blossoms were emerging from the buds (after sporodochia had appeared) and found that the trees developed markedly less blossom infection than unsprayed trees. Wilson (1950) applied an aqueous solution of sodium pentachlorophenate to apricot and almond trees as the unopened flowers emerged from the winter buds; many of the conidia present on the trees were destroyed and only a few new ones were produced before the end of the blossoming period.

PRE-BLOSSOM SPRAY. G. H. Berkeley (1928) found that a pre-blossom spray of wettable sulphur was very effective in controlling blossom blight of peaches and later, in 1929, also recommended a pre-blossom application of sulphur dust or lime sulphur. Hurt (1937) recommended lime sulphur solution on one of the wettable sulphurs to be applied as the pink colour begins to show in the peach buds. Rudolph (1936) advised that apricots and peaches should be sprayed with Bordeaux mixture. Folke (1950) tested a number of the synthetic organic fungicides at the pre-blossom stage with promising results; Johnson (1950) in pre-blossom spraying on acid cherries obtained good control with Puratized Agricultural Spray 1 pt. to 100 gal. and the Fungicide Committee of the American Phytopathology Society (1949) reported that Krenite applied at the pop-corn stage destroyed 60 per cent of the bloom but that a larger and cleaner crop was harvested, blossom blight being reduced from 84 per cent in the untreated trees to 8 per cent in the treated.

BLOSSOM SPRAYS applied when some of the flowers are open or even at full bloom. When it was found that the flowers of deciduous fruit trees of temperate regions could be sprayed with little or no injury, another method of controlling the blossom blight phase of brown rot was suggested and many trials have been conducted to determine the best period during flowering to apply sprays and the best fungicide.

Howard and Horne (1921), from observations on apricots, concluded that protection was excellent when spraying was done at the time the trees were coming into bloom. They found that Bordeaux mixture 4:2:50 was a reliable spray but that some injury was caused by sulphur sprays. Hammond (1923) in Australia recorded perfect control on cherries when 10 per cent or 50 per cent of the flowers were open. The application of 5:5:50 to 8:8:50 Bordeaux mixture on cherry trees as the first blossoms open gives good control except when rain occurs at harvest time (Philp, 1930); Calavan (1944) obtained good results on cherries with Bordeaux mixture 3:4:50 as a blossom spray after an eradicant spray. Petersen and Cation (1949) sometimes found severe burning of cherry petals after spraying in partial or early bloom with liquid lime sulphur 2 gal. to 100 gal. The browning was most severe when applications were made between 10 a.m. and 4 p.m. but sprays applied later than 4 p.m. caused less burning. The damage was most severe on the north side of the trees. No such damage was observed on peach blossoms in the same areas and with the same spray. Later (1951) they recorded good results with wettable sulphur 5 lb. in 100 gal. water and with liquid lime sulphur 2 gal. to 100 gal. applied when 25 per cent of the blossoms were open, followed by a second spraying when all the blossoms were open.

Sharvelle and Burkholder (1947) in their Purdue (Indiana) spray schedule recommended using wettable sulphur when 25 per cent of blossoms were open.

Promising results from blossom spraying with some of the organic synthetic fungicides have been obtained, e.g., Yarwood (1948) with 0·2 per cent Fermate and spreader on apricot at full bloom, Foster (1949) and Powell (1951) with phygon on peach.

POST-BLOSSOM APPLICATION. The petal-fall and shuck (the withered calyx)-fall stages, are also mentioned by some investigators as suitable periods for the application of fungicides to prevent early infection of the fruit.

C. F. Smith and Clayton (1950) after recommending spraying peaches in bloom say that at the petal-fall stage a fungicide is not often needed but as an insurance, wettable sulphur is added to the petal-fall and to the shuck sprays.

Sherbakoff and Andes (1936) recommended a petal-fall spray and one or more pre-harvest applications of sulphur after July if conditions were favourable for brown rot of the fruit.

SUMMER OR COVER SPRAYS applied during the development and maturation of the fruit, the preparations being mostly preventive though possibly at times they have a direct fungicidal action on conidia falling upon or developing on the surface of the fruit.

Fungicides used as cover sprays are numerous and very varied though usually they are sulphur-containing sprays or dusts. Some of the synthetic organic compounds have given promising results but have not yet come into general orchard practice.

Several applications are generally recommended, starting soon after the petals have dropped and repeated at intervals of ten days to a fortnight until the pre-harvest stage. The later and the pre-harvest sprays are applied chiefly with the object of protecting the fruit from brown rot after it is picked, stored and put on rail.

Britton *et al.* (1922) found dusts to give slightly healthier fruit than sprays but to be more costly. Anderson (1933) and Sauchelli (1933) recommended flotation sulphur (average particle size about 3µ).

Lime sulphur (2–3 qts. per 100 gal.) is said to be less effective than wettable sulphur or sulphur dusts applied several times during the season for post-harvest control (Weaver, 1948). Winter and Young (1938) recommended a dry-mix home-made sulphur preparation as being less expensive than wettable sulphur; one application of either made three weeks after calyx-fall kept infection down to an average of 1·5 per cent, the unsprayed controls averaging 95 per cent fruit infection.

Synthetic organic preparations mostly used as cover sprays have already been mentioned (see p. 50).

PRE-HARVEST (PRE-PICK) SPRAYING. A spray or dust may be applied shortly before the fruit is picked to protect the ripening or mature crop. Sulphur cover sprays put on late are usually employed but here again, as with cover sprays, there is no agreement as to the best times of application or of the most suitable preparation to be used as will be seen from the examples mentioned below.

Dutton and Johnston (1922) said that a sulphur dust (sulphur hydrated lime) applied three days to a month before picking greatly improves the keeping qualities of the fruit but that such treatment should only be considered as supplementary to the regular summer schedule. Winter and Young (1938) stated that a pre-harvest application was very important against brown rot. Hurt (1935) got the best control by a pre-harvest treatment with either flotation sulphur or a new pink sulphur blending with the colour of the fruit and applicable as a spray or dust. Hessler (1942) recommended a fine grade sulphur dust applied three weeks before starting to pick and again after ten days with another application if rot shows. Poulos (1950) applied Sulfuron X (5–100) as a pre-harvest spray. In Australia, Fish (1933), and Anon. (1945), recommended ammonium polysulphide for applications near time of picking, as it leaves no noticeable deposit on the fruit. Palmiter and Hamilton (1942-45) tried, with some success on ripening cherries, the newer organic preparations, Fermate, Jap Beetle Spray and Isothan Q15.

Rudolph (1936) considered that spraying stone fruits immediately before picking was inadvisable, particularly for canning fruit but that an application should be made three or four weeks before gathering whether brown rot was present or not. Stevenson (1926) found that lime sulphur sprays applied a few days before picking and canning may cause " hydrogen springers " from the liberation of hydrogen and the consequent loss of vacuum in the cans. Culpepper and Moon (1920) had previously remarked that, since the adoption of sulphur sprays for the control of brown rot on peaches for canning, difficulty had been encountered because of the swelling of the cans a few months after canning, and attributed it to the formation of hydrogen sulphide and hydrogen gas derived from the sulphur adhering to the fruit.

POST-HARVEST TREATMENT during storage and transport. After the fruit is picked, elementary precautions—sterilizing the cases (with formalin (Cunningham, 1922*d*) or with hot water (Henrick, 1935)) and storehouses and employing cold storage—are advised but for period storage and transport, these measures have to be supplemented by others.

Orchard practice during summer has a great effect on the amount of rot in storage. Brooks and Fisher (1921) concluded that sweet cherries from orchards in which there was less than 1 per cent of rot on either sprayed or unsprayed at picking, later developed 24·3 per cent on fruit from unsprayed trees and only 6·4 per cent on fruit from sprayed trees; the corresponding figures for Italian prunes were 28 per cent and 7·1 per cent.

For preventing loss in the cases, various measures have been tried such as dipping tissue wrappings (Willison, 1944), metabisulphite tablets (Willison, 1944), iodized fruit wrappings (Manley, 1947) and applying CO_2. In dipping tests by Heuberger *et al.* (1949, 1950) liquid lime sulphur was the only chemical which gave appreciable control. The New Zealand D.S.I.R. recorded (1944) that Shirlan, as a dip for apricots, gave a high degree of control. A special " F.M.C. Stericooler," with ice water containing Hypochlor was described by Cardinell and Mitchell (1948). Heat and moisture are factors of special importance in transport; heat relations have been carefully studied (English & Gerhardt, 1942) and refrigerator cars with fans were recommended by Rose *et al.* (1950), who said that in end-bunker refrigerator cars not equipped with fans, brown rot is usually worse at the top of the load than at the bottom. Bratley and Wiant (1950) advised prompt and thorough refrigeration for retarding rot of peaches and plums.

The use of CO_2 has been studied by a number of investigators. Brooks *et al.* (1932, 1936) stated that within the range of 10–50 per cent of CO_2, the average reduction in brown rot infection was approximately twice that of the percentage of gas used and that cherries exposed to an atmosphere containing 30 per cent CO_2 had less decay and were firmer and fresher than similar fruit held at the same temperature without exposure to CO_2.

Fungus decay (including brown rot) was controlled during seventeen to twenty days storage at 45° F. in an atmosphere containing 25 per cent CO_2

(Gerhardt & Ryall, 1939). Cherries sent under simulated transport conditions from California in an atmosphere containing CO_2 arrived at eastern markets in better condition than fruit sent in cars without the CO_2 supplement to refrigeration. This was recorded by English and Gerhardt (1942) who added that the inhibition of brown rot by CO_2 at concentrations within the range encountered in a commercial shipment of sweet cherries was clearly shown.

Shutak and Christopher (1948) found that CO_2 retarded ripening, decreased the spread of brown rot and prolonged the "shelf life" of plums.

Treatment of Apothecia

In America and other countries where *Sclerotinia fructicola* abounds, the development of apothecia on fallen fruit and the ejection of ascospores in spring is recognized as a real danger and orchard sanitation should include the ploughing-in of fallen mummified fruit before the blossoms open (Rudolph, 1936).

Chemical methods of preventing infection from ascospores have also been tested. It has been shown that calcium cyanamide applied as a dust to the soil in orchards prevented the emergence of the apothecia (Huber & Baur, 1939*a*; Baur & Huber, 1940).

Trials and Recommendations in Europe

In Europe it has not been found necessary to devise elaborate programmes like those recommended against *Sclerotinia fructicola* on stone fruits in America.

The apothecia of *S. fructigena* and *S. laxa* are so rare that precautions against infection from ascospores are unnecessary, but eradicant sprays, to destroy the sporodochia developing from the overwintering mycelium in mummified fruits, in spurs, and in twigs and branches, have been tested, and sometimes recommended, particularly for the control of blossom wilt caused by *S. laxa.*

A mixture of caustic soda and soft soap (1 lb. of each in 100 gal. water) was found to destroy, or render sterile, the sporodochia of *Sclerotina laxa* forma *mali,* but orchard trials did not yield conclusive results, and this wash has not come into general use, though Jørstad (1932) recommended 1 per cent caustic soda with green soap against brown rot of cherries in Norway.

The caustic soda washes have been superseded by tar oils as eradicant sprays. Lees and Briton Jones (1925) with 7 per cent "Carbo fluid" completely controlled brown rot on plum trees. Moore *et al.* (1939) found a 10 per cent tar oil spray applied in January assisted in controlling blossom wilt on Morello cherries. Hus (1933) recorded favourable results with carbolineum against *Monilia* on cherries in Holland, as also did Estienne and Soenen (1946), and Schenk (1951) in Belgium.

Marsh (1947) and Byrde (1949) in small-scale tests found phenyl mercury chloride to be effective against the sporodochia on mummified apples and plums, and Byrde (1950) also recorded promising results with sodium and calcium arsenite preparations, sodium dinitro-*ortho*-cresolate and sodium pentachlorphenate.

Spraying cherries when in blossom with lime sulphur has been found effective by at least one grower in north Kent, though some varieties are more sensitive to this spray than others, and the necessary precautions should be taken.

Brož (1913) advocates spraying with 1 per cent Bordeaux mixture before or during flowering to control blossom wilt.

The spray schedule of Anrès and Joëssel (1932) for apricots in the Rhône valley was: one application when the buds begin to burst, a second about the time of blossoming and a third when the withered perianth drops from the young fruit (shuck-fall).

Youganova (1946, 1951) elaborated a copper naphthanate spray for use in the Black Sea coastal regions of Russia where disastrous outbreaks of apricot blossom wilt, caused by *S. laxa,* have occurred (see p. 12). Her control measures involved (1) spraying with 6 per cent emulsion containing 0·6 per cent naphthanate in the "pre-winter" period, (2) 6 per cent emulsion containing 0·6 per cent copper naphthanate in early spring up to the time the buds begin to swell, (3) 3 per cent emulsion containing 0·3 per cent just before bud-burst, (4) 1 per cent emulsion when the shuck is thrown off. In 1950 Youganova and Sushitski reduced the number of applications to three for apricots, and sweet and acid cherries: (1) when the buds begin to swell, (2) before the flowers burst, (3) when the first flowers are open, the three sprays to be in decreasing concentrations. The compound consists of copper sulphate and naphthalene soap combined by an emulsifying process using oil and kaolin, and its subsequent dilution to the required concentration. It should be noted that Byrde (1949) in his tests with apple and plum mummies bearing sporodochia of *S. fructigena* found copper naphthanate of little use as an eradicant spray.

Ludwigs (1928) says that twig blight in Germany may be combated by two applications (dormant and spring) of 3–4 per cent Bordeaux mixture, while fruit rot is preventable by spraying with 1 per cent Bordeaux mixture when the cherries begin to form.

Rosella (1939) stated that Monilia on apricots in France may be controlled by copper-containing sprays applied (1) just before the renewal of growth, (2) immediately prior to the opening of the flower buds, (3) after blossoming.

Estienne and Soenen (1946) in Belgium reduced the degree of infection on acid cherries by (1) spraying in winter (early March) with carbolineum and with a spray fluid containing dinitro-*ortho*-cresol, (2) a pre-blossom spraying with copper oxychloride and with an organic sulphur preparation.

The repeated cover sprays, found to be so necessary against peach brown rot in America, are not practised against fruit brown rot in Europe. Attempts have been made to prevent brown rot on apples by summer spraying, hitherto with little success (see p. 64). Byrde (1949), with a late June spray of a phenyl mercury chloride preparation, obtained approximately three-quarters reduction in the current season's brown rot infection of plums in a small-scale trial; lime sulphur gave less control.

The prevention of post-harvest rot, other than by hygienic measures, has received but little attention in Europe. Meier and Grampoloff (1936) studying the effect of ultra-violet irradiation on stored fresh fruit record that the invasion of peaches by "Monilia" was reduced by treatments for 5 min. 40 sec. each day for periods of 3 to 6 weeks, but there is no evidence that this is of practical use.

HOST PLANTS

The three brown rot fungi here described are responsible for great reduction in the yield of fruit trees of species included in the suborders Pomoideae and Prunoideae of the family Rosaceae. All the commonly cultivated fruits and many kinds of ornamental shrubs of the genera *Pyrus* and *Prunus* are subject to brown rot infection. Sometimes species of other rosaceous genera are affected and (but rarely) representatives of other genera.

Unusual hosts have frequently been mentioned. E. F. Smith (1889) wrote that it had been reported on grapes, gourds, the medlar and *Cornus mas*, but this appears to be a reference to a note by Sorauer (1897) and so does not concern the American brown rot fungus. He also says that Dr. Arthur induced it to grow on blackberries ; the fungus in this case was probably *Sclerotinia fructicola.* Reade (1908) collected "*Sclerotinia fructigena*" (?*S. fructicola*) on the ripe fruit of *Prunus virginiana* L., apparently growing as a saprophyte.

In inoculation experiments with *Sclerotinia fructicola* Norton (1914) obtained successful infection with the development of conidia, on cherry, wild goose plum, blackberry, Japanese plum, damson plum, green corn, pear, peach, apple, *Prunus serotina,* strawberry, crab apple, *Crataegus, Pyrus betulifolia,* dried prune, peach, apricot and raisin. About 50 species of many families, including *Rosa, Amelanchier, Rubus occidentalis, Pyrus arbutifolia,* and *Cydonia japonica,* showed no outward sign of infection ; some of these, however, are included in Roberts and Dunegan's list mentioned below.

In discussing the host-range of *Sclerotinia fructicola* in Australia, Harrison (1928) recorded that he had isolated the brown rot organism from the fruits of apple, apricot, blackberry, cherry, nectarine, peach (commercial), peach (ornamental), pear, plum (English and Japanese and prune), and quince from orchards in New South Wales. He also infected artificially the fruits of grape, loquat, persimmon and tomato.

Roberts and Dunegan (1932) says that under favourable conditions the fungus [*S. fructicola*] is probably able to infect all drupaceous and pomaceous species as well as many other members of the Rosaceae, and they give a list of 41 hosts on which it has been found ; these are mostly species of *Prunus* and their hybrids, but flowering quince (*Chaenomeles japonica*), rose, blackberry and blackcap (*Rubus occidentalis*) are included.

With reference to the less commonly cultivated rosaceous hosts, mention should first be made of the pomaceous fruits quince and medlar which are frequently grown in European gardens, and of the varieties of plum included under *Prunus insititia* and the myrobalans or cherry plums, *P. cerasifera* (*divaricata*), which are also sometimes grown for their fruit.

Quince (*Cydonia vulgaris*) fruits are often attacked in Europe by *S. fructigena,* rarely by *S. laxa.* On Illinois markets quince fruit rot caused by *S. fructicola* is stated to be common and destructive (Fischer, 1930).

On medlar (*Mespilus germanica*) infection of the fruit by *S. fructigena* has been recorded in Kent (Wormald, 1939) and *S. laxa* has once been found on a young fruit and on a leaf. Medlar, however, appears to be a very unusual host for the true brown rot fungi, though it is very susceptible to one of the leaf blotch Sclerotinias (see p. 22).

Varieties of *Prunus insititia* (damsons, bullace and the St. Julien plum) and forms of *P. cerasifera* are subject to infection similar to that in the more commonly cultivated commercial plums (varieties of *P. domestica*), but the fruit of *P. insititia* appears to be particularly susceptible to infection by *S. fructigena*; on one occasion 50 per cent infection of the fruit on a St. Julien plum tree in a garden was observed (Wormald, 1940).

Attacks on a number of ornamental rosaceous shrubs have been observed in Great Britain and on the Continent, mostly as blossom wilt (accompanied by the killing of the flower-bearing twigs) caused by *S. laxa*; but *S. fructigena* is to be found on a few fruits.

The writer has seen *S. fructigena* on the fruit of *Pyrus purpurea* from three localities in the south of England. On the Continent the same fungus is recorded as infecting the fruit of *Pyrus baccata*, in Germany by Linsbauer (1925) who found fruit heavily attacked in 1923, and in Poland by Garbowski and Juraszkówna (1933).

In the south of England *Monilia fructigena* often infects the fruits of ornamental almonds (*Prunus amygdalus*), and sometimes clusters of the mummified fruits are conspicuous after leaf-fall.

In Bulgaria, *Monilia fructigena* is recorded by Christoff (1934*a*) as occurring on *Prunus hortulana, P. pissardii, P. spinosa* and *P. divaricata* [= *P. cerasifera*], and *Monilia cinerea* on *Prunus insititia*.

Jaap (1908) mentions *Sclerotinia cinerea* as occurring on the fruit of *Prunus spinosa* in Austria.

In Japan *Sclerotinia fructigena* is said to appear on the ripe fruits of *Prunus mume, P. pseudo-cerasus*, grape and *Elaeagnus macrophylla* (Takahashi, 1911).

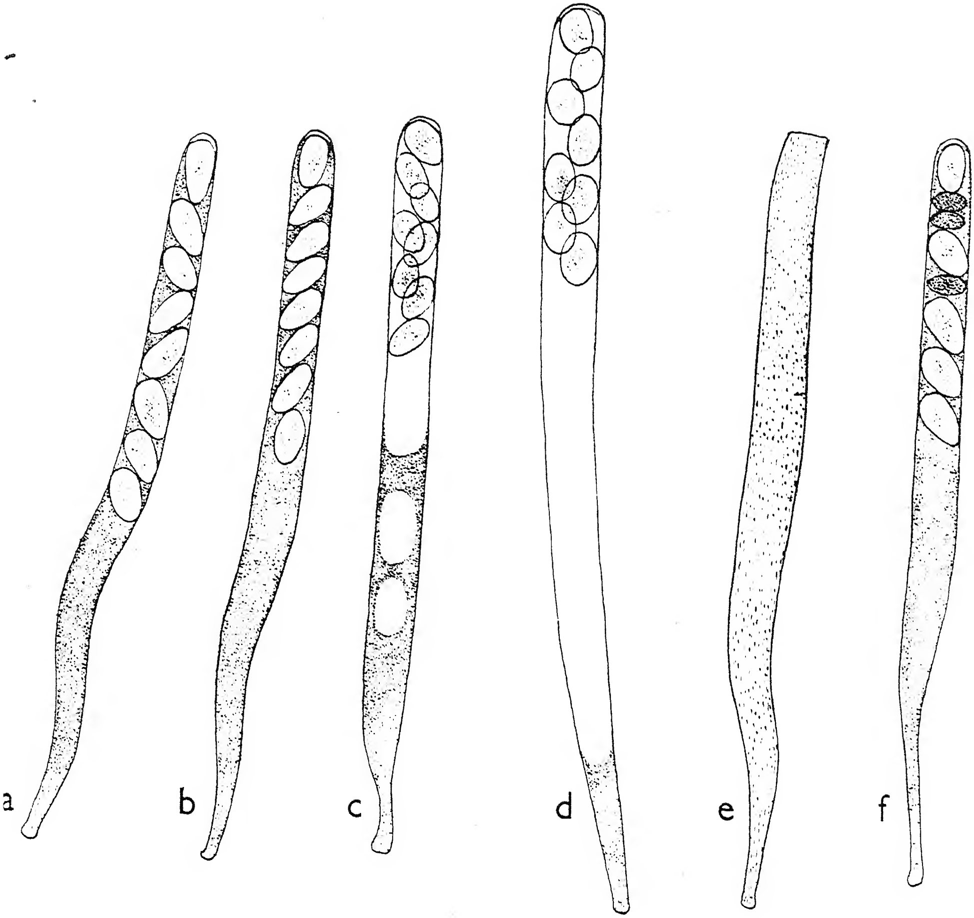
With regard to blossom infection, some ornamental rosaceous shrubs infected by *Sclerotinia fructicola* are included in the list of host plants given by Roberts and Dunegan (1924). In Europe those that have been found infected by *S. laxa* may be listed as follows:

Host	*Country*	*Reference*
Pyrus aria	England	Wormald, 1940
P. elaeagrifolia	,,	,, ,,
P. japonica	,,	,, ,,
P. purpurea	,,	,, ,,
Prunus besseyi	Scotland	Dennis & Foister, 1942
	England	Wormald (unpublished)
P. nana	Denmark	Gram & Weber, 1940
	England	Wormald, 1940
P. padus	,,	,, ,,
	Scotland	Dennis & Foister, 1942
*P. pseudocerasus**	Denmark	Weber, 1926
P. pumila	England	Wormald, 1940
P. salicina	France	Evreinoff, 1943
P. serrulata	England	Wormald, 1940
P. tomentosa	,,	,, ,,
P. triloba	Denmark	Weber, 1926
	Sweden	Lindfors & Holmberg, 1941
	Norway	Jørstad, 1945

---

* Ezekiel (1924*b*) mentions receiving from Holland a strain isolated from this host.

Details of observations in England mentioned above are given by Wormald (1940) except for *Prunus besseyi*. On this host infection was observed in the spring of 1942 on some seedling plants at the East Malling Research Station. In May several of the plants showed withering of the main stem or of branches (one-year-old twigs); on these there were wilted flowers bearing Monilia fructifications on the flower stalks. The fungus isolated proved to be *Monilia cinerea* and it gave typical infection when inoculated into plums.



[Drawing: *Annals of Botany*

Asci of *Sclerotinia laxa* [× 500]

a and *b*. With monostichous spores

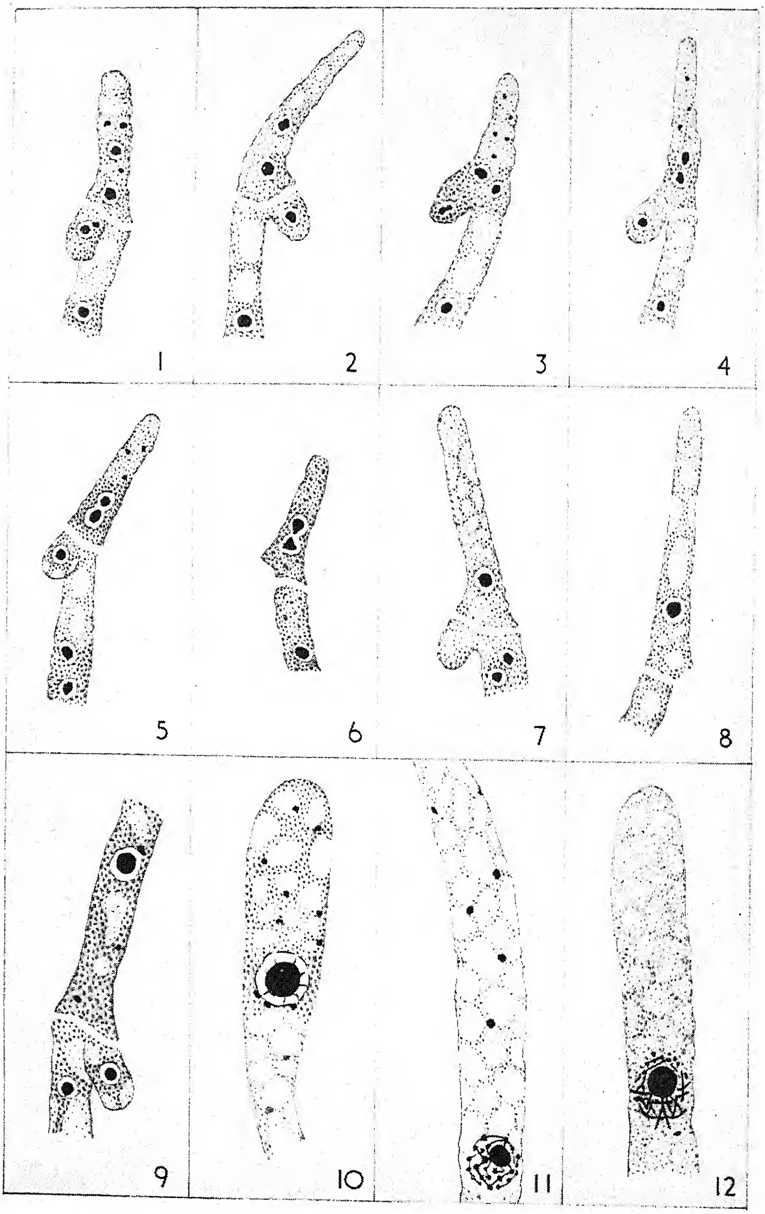
c. An ascus becoming vacuolate; spores not strictly monostichous

d. An ascus with a single large vacuole and distichous spores; condition a few seconds before the ascus dehisced

e. The same ascus as *d*, after dehiscence

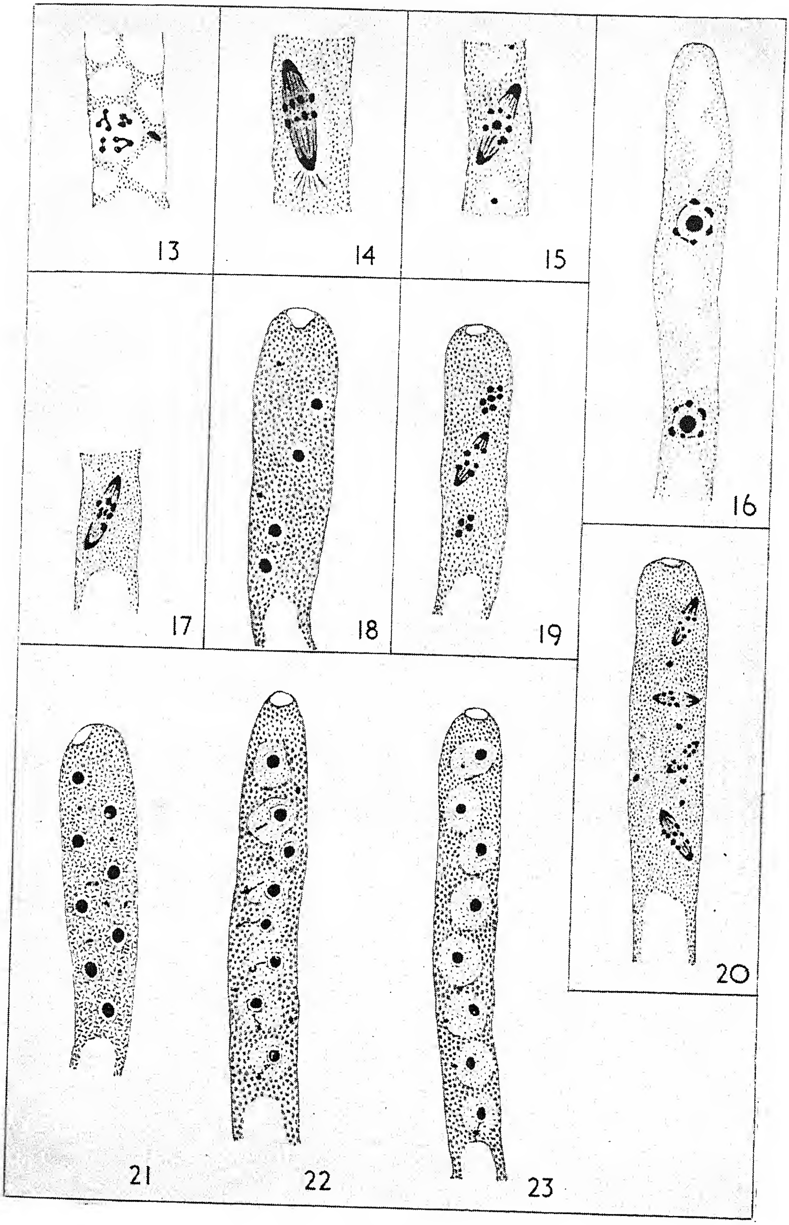
f. An ascus with three abnormally small spores

PLATE I



Ascus Development . . .

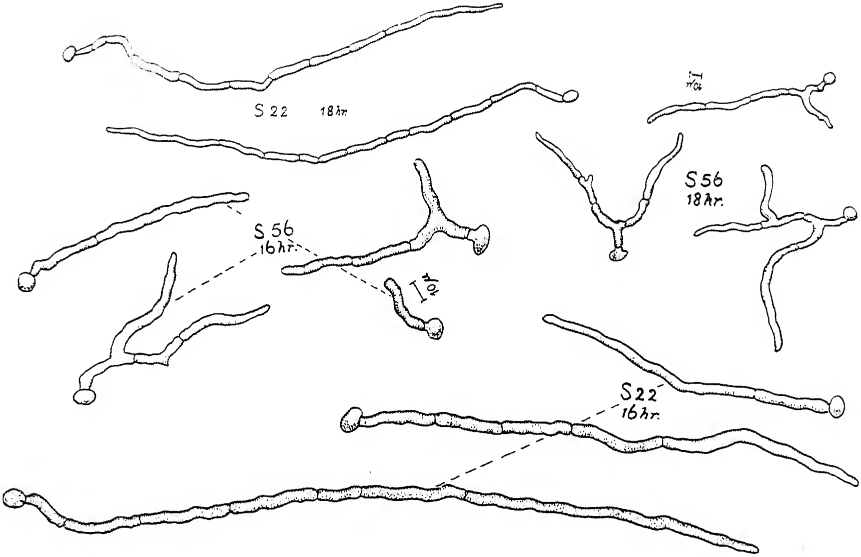
PLATE II



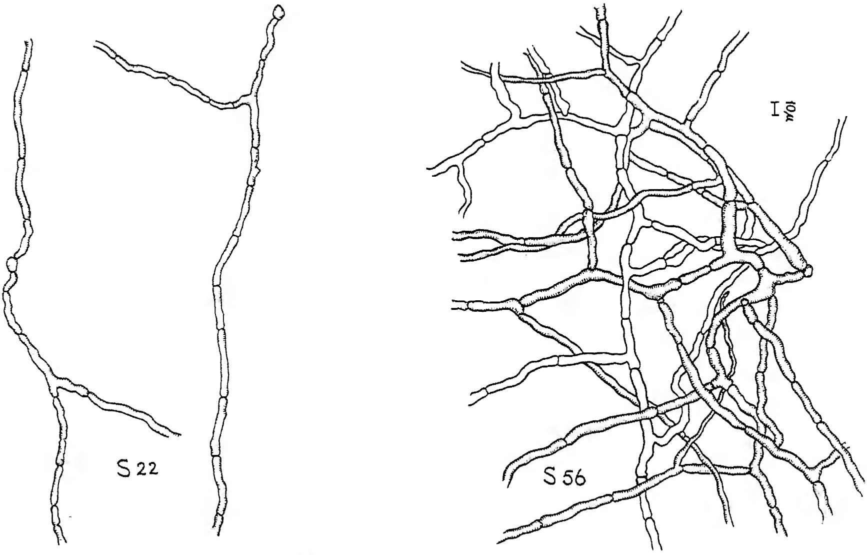
. . . In *Sclerotinia fructicola*

(For descriptive details see Heuberger, 1934)

PLATE III



A. Conidia of *Sclerotinia fructicola* (S 22) and *S. laxa* (S 56) germinating in drop cultures of potato dextrose deccction at 25° C. Note the characteristic geniculate branching in S 56

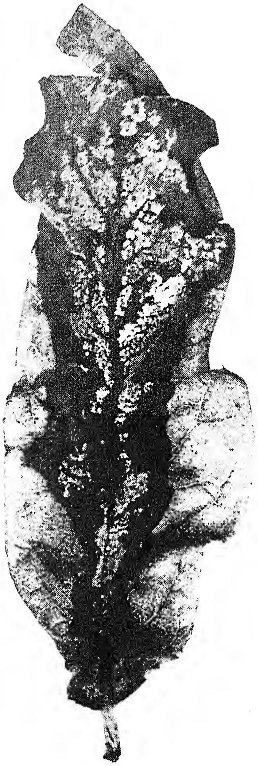


B. General Mycelial Characteristics in 2-day-old Drop Cultures of *S. fructicola* (S 22) and *S. laxa* (S 56)

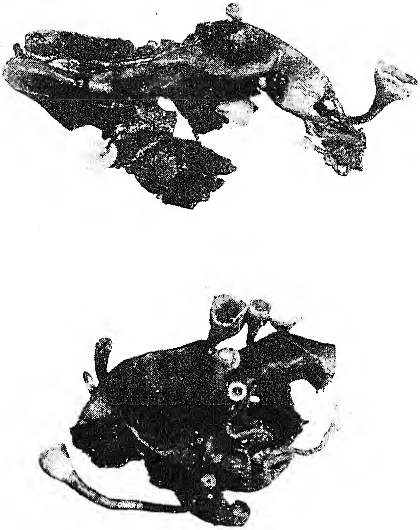
Growth is more extensive with *S. fructicola* but very loose; with *S. laxa* a great amount of branching occurs though the entire colony is small

(After Ezekiel)

PLATE IV

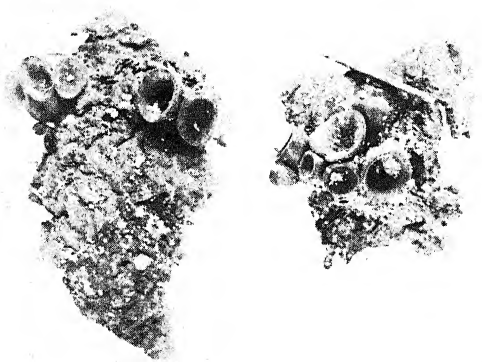


A. Leaf Blotch of Medlar showing conidial fructifications of *Sclerotinia mespili*



[*Photo: Annals of Botany*

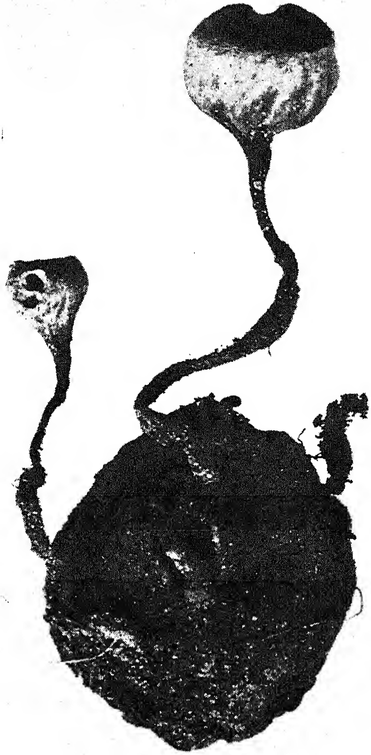
B. Apothecia of *Sclerotinia laxa* growing out from mummified plums



[*Photo: U.S. Dept. of Agriculture*

C. Apothecia of *Sclerotinia fructicola* developing from two partly mummified peaches

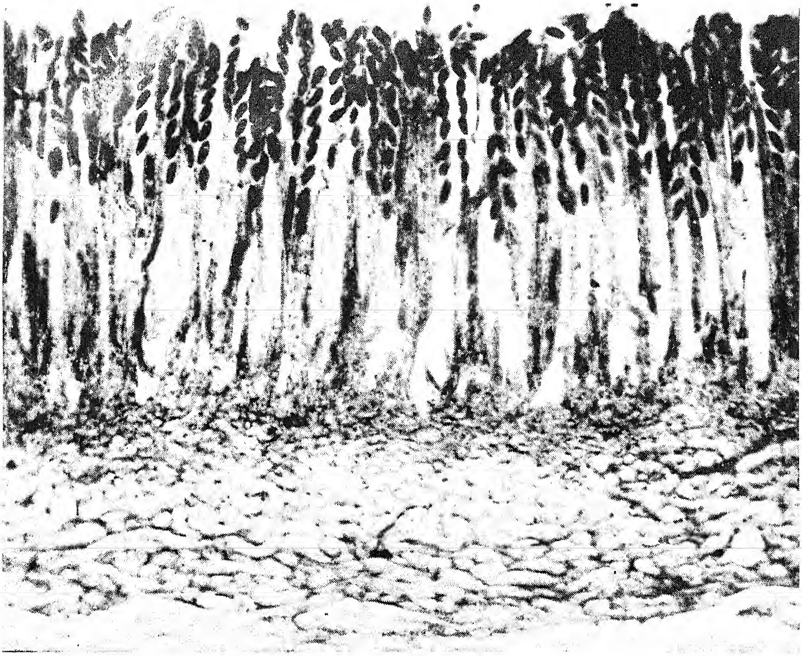
(From U.S.D.A. Tech. Bull. 328)



[*Photo: G. C. Wade*

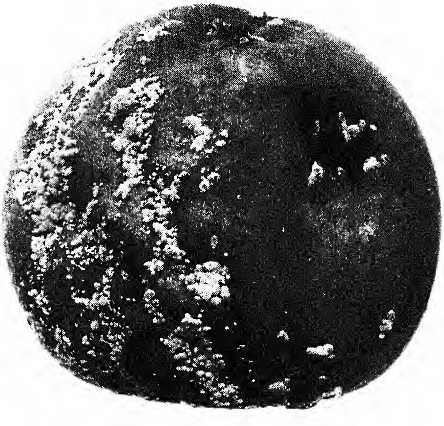
D. Apothecia of *Sclerotinia fructicola* on a mummified apricot

PLATE V

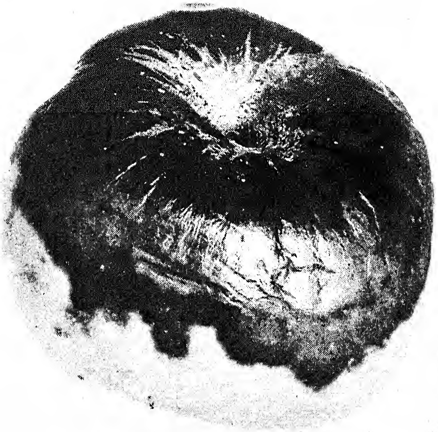


[*Photo: U.S. Dept. of Agriculture*

A. ASCI OF *Sclerotinia fructicola*
(From U.S.D.A. Tech. Bull. 328)

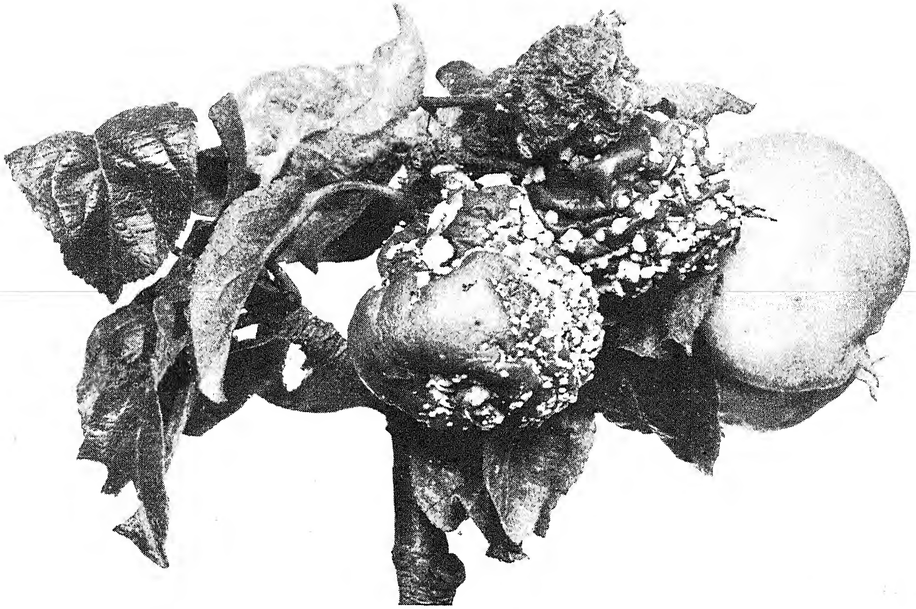


B. BROWN ROT INFECTION OF APPLE
through a scab lesion

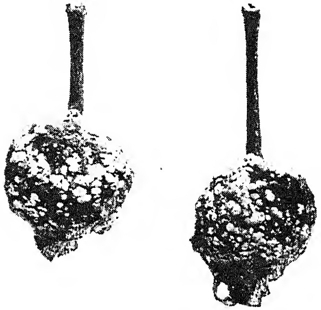


C. BROWN ROT INFECTION OF APPLE
through the wound left by picking the frui
without its stalk. A Grenadier apple four day
after it was picked

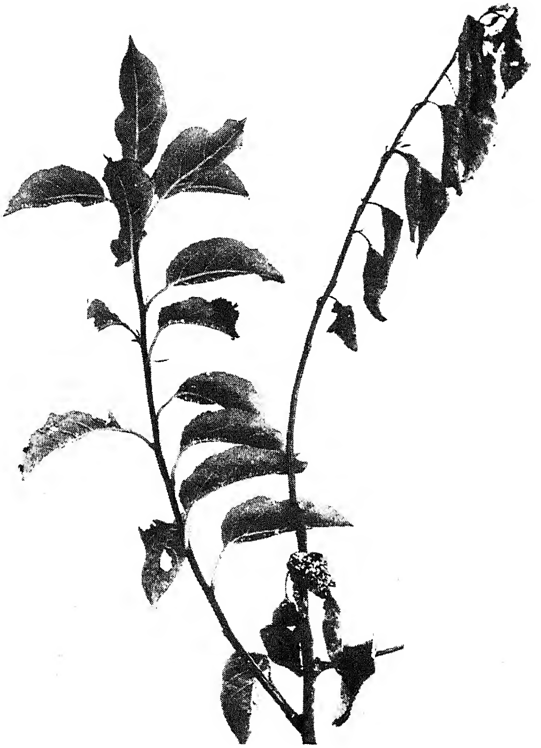
PLATE VI

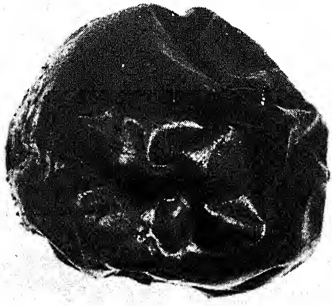


A. Brown Rot of Apples caused by *Sclerotinia fructigena* showing infection extending from one apple to others in contact



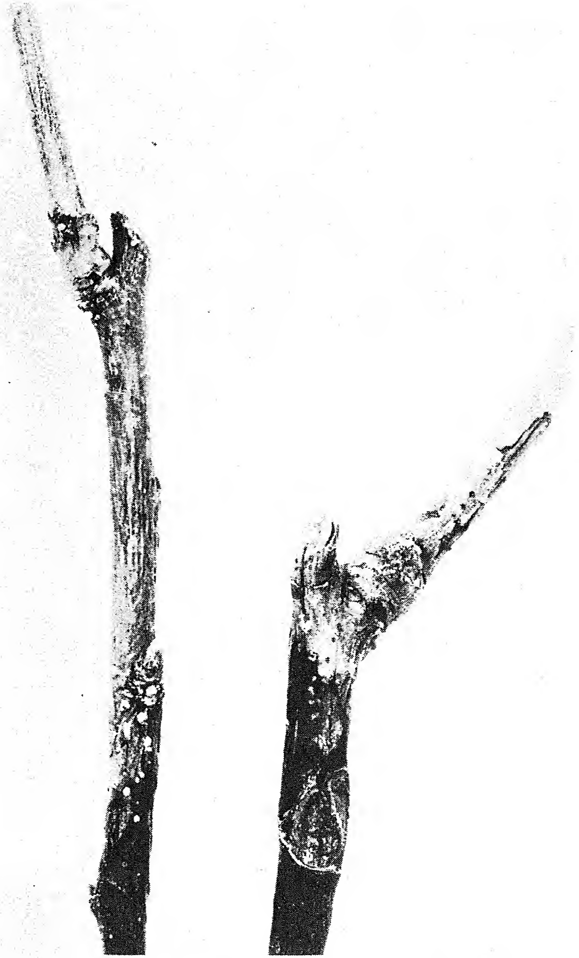
B. *S. fructigena* on Young Apples





C. Black Apple caused by *S. fructigena*

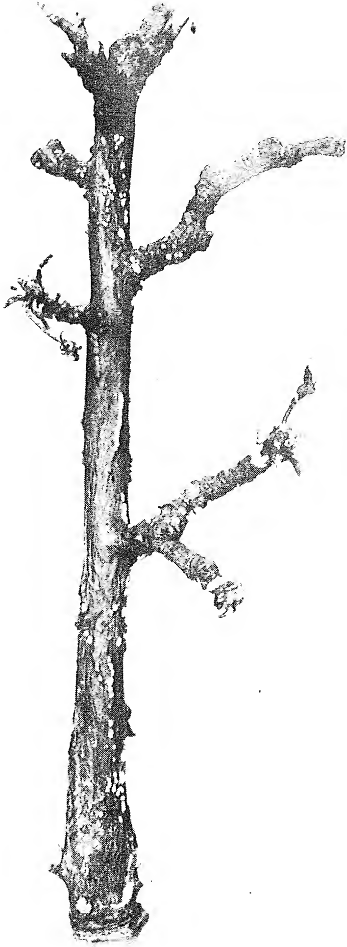
D. Apple Canker caused by *S. fructigena*. Infection started in the fruit and extended into the branch



A. Apple Cankers

showing Monilia fructifications of *S. fructigena* developing during the summer following that in which infection occurred

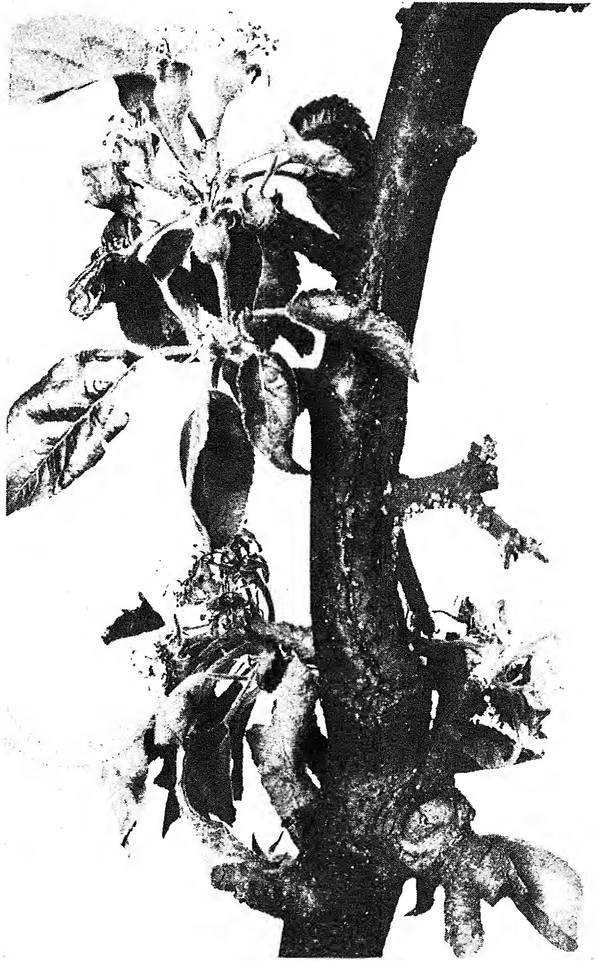
[Photo: Brit. Mycol. Soc.



B. Brown Rot Canker

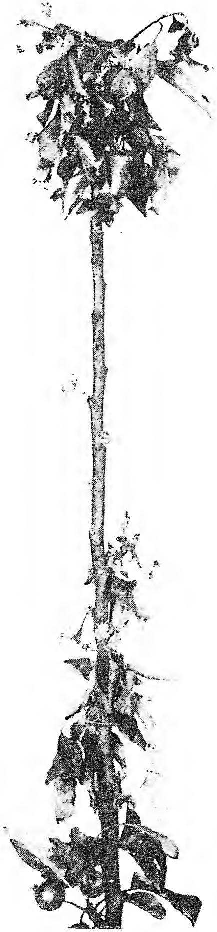
(following Blossom Wilt) in the spring, showing numerous Monilia fructifications of *S. laxa* f. *mali*

PLATE VIII



A. Blossom Wilt of Apple

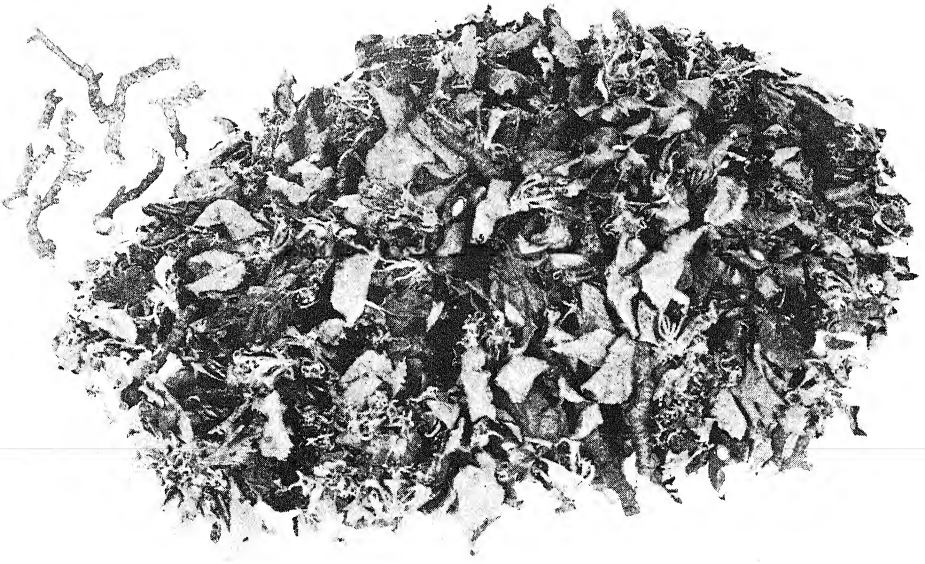
showing a canker with a dead spur, and below it wilted inflorescences



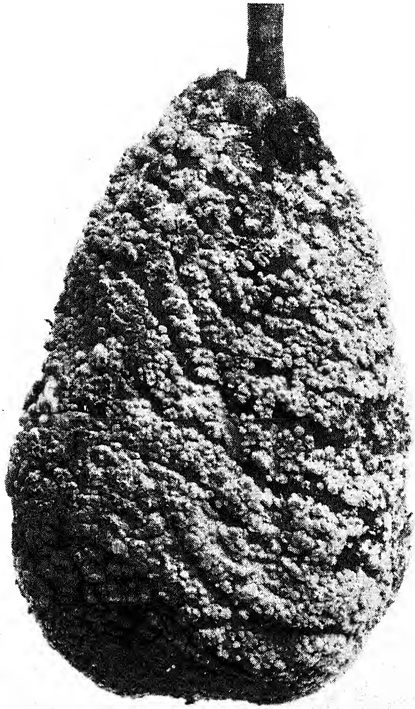
B. Stem of a Cordon Apple Tree

(var. Rival) showing upper part killed by a canker following Blossom Wilt

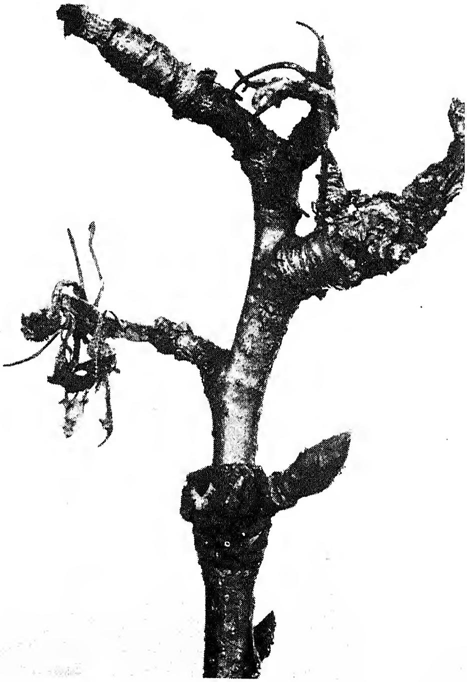
PLATE IX



A. Heap of Dead Apple Flower Trusses
(211 in number) cut from 10 trees that bore the 7 dead spurs with Monilia fructifications shown on the left



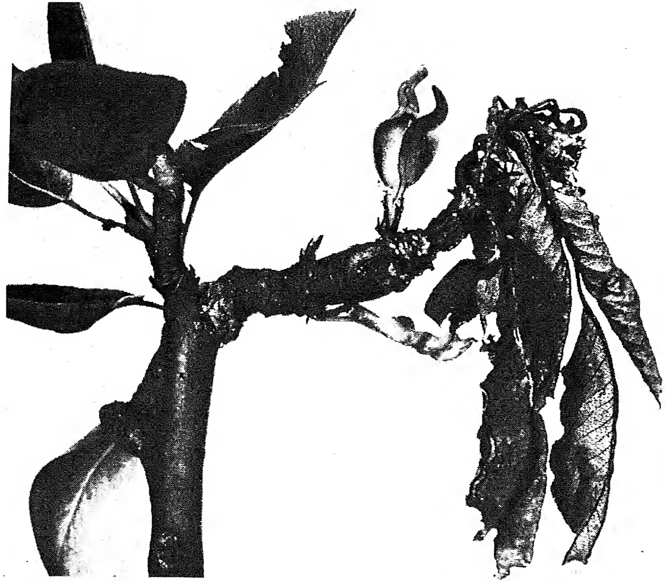
B. Brown Rot of Pear
(*Sclerotinia fructigena*)



[*Photo: Annals of Botan*

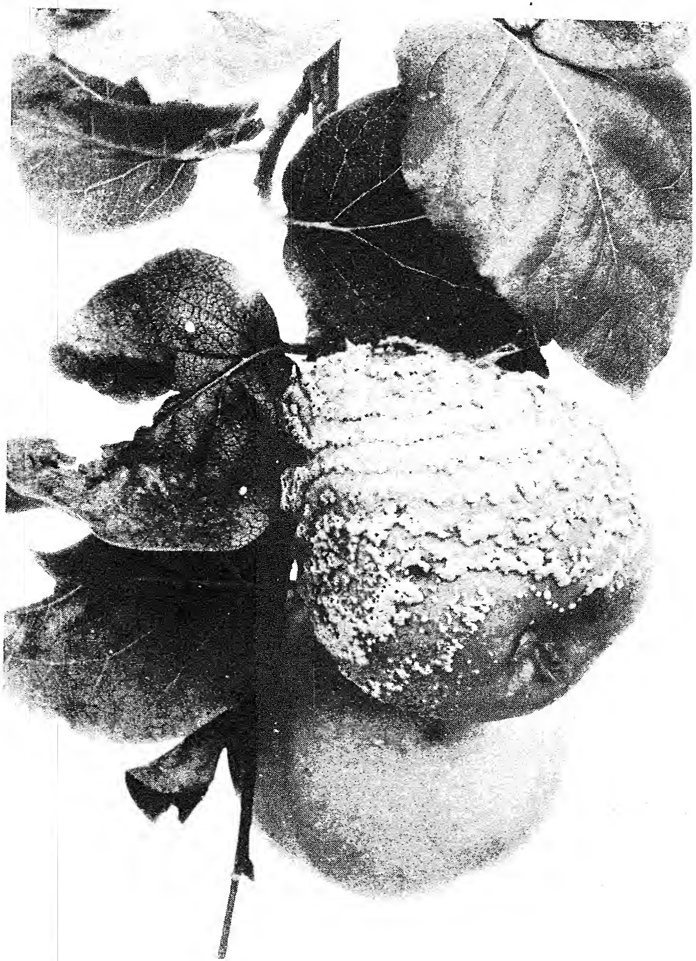
C. Blossom Wilt of Pear
Winter condition

PLATE X



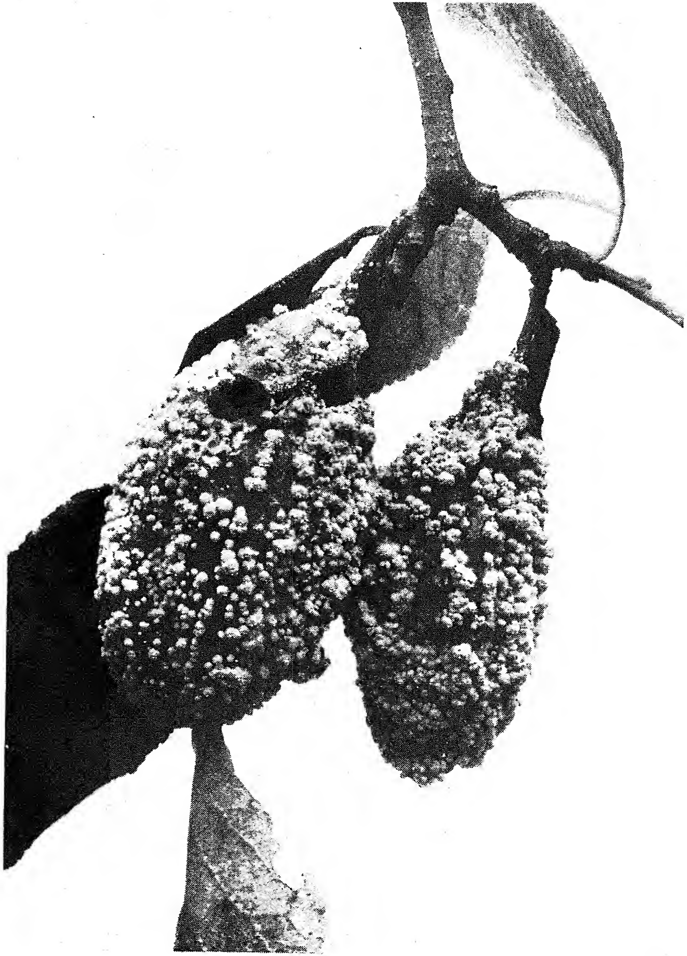
[Photo: *Annals of Botany*

A. Blossom Wilt of Pear
(*Sclerotinia laxa*)
Summer condition

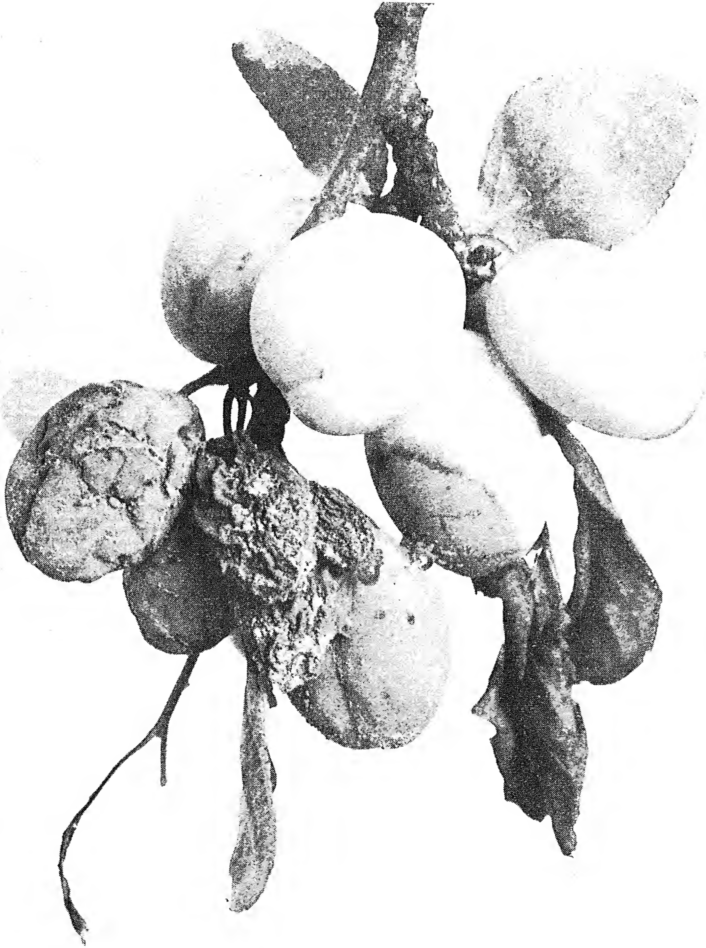


B. Brown Rot of Quince
(*S. fructigena*)

PLATE XI

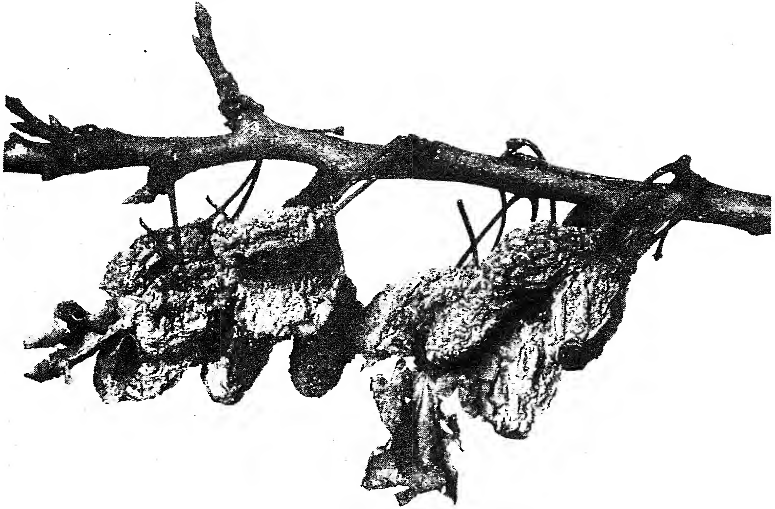


A. PLUMS WITH *Sclerotinia fructigena*

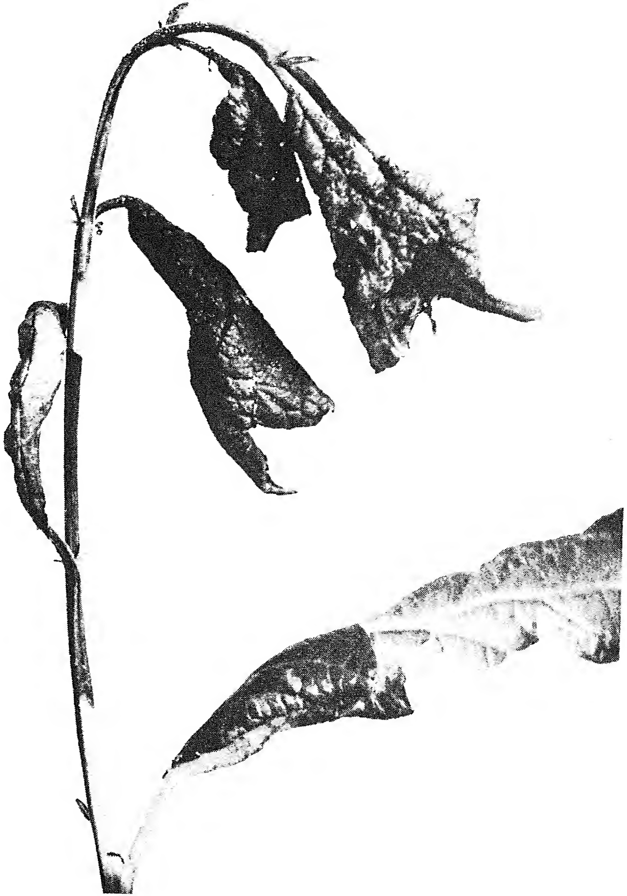


*B. PLUMS WITH *Sclerotinia laxa*

[*Photo: Annals of Botany*



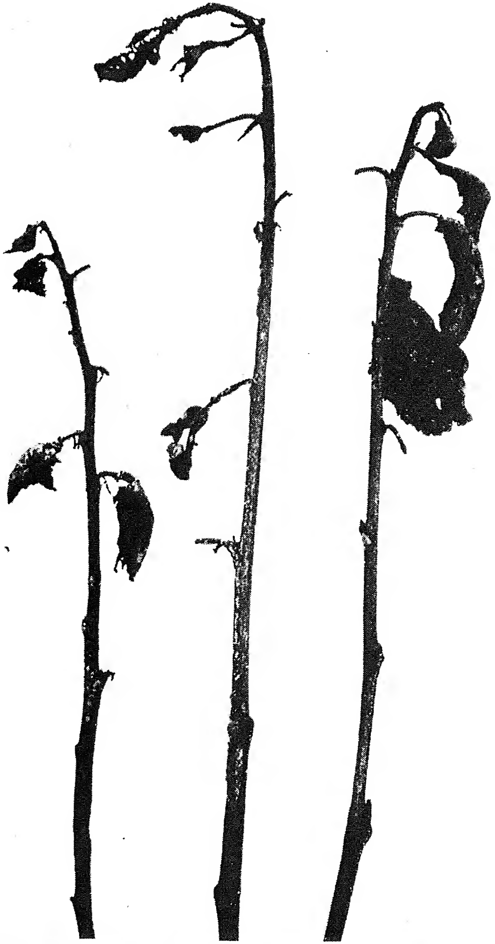
A. Mummified Plums (with *S. laxa*) in Winter



B. Wither Tip on Victoria Plum

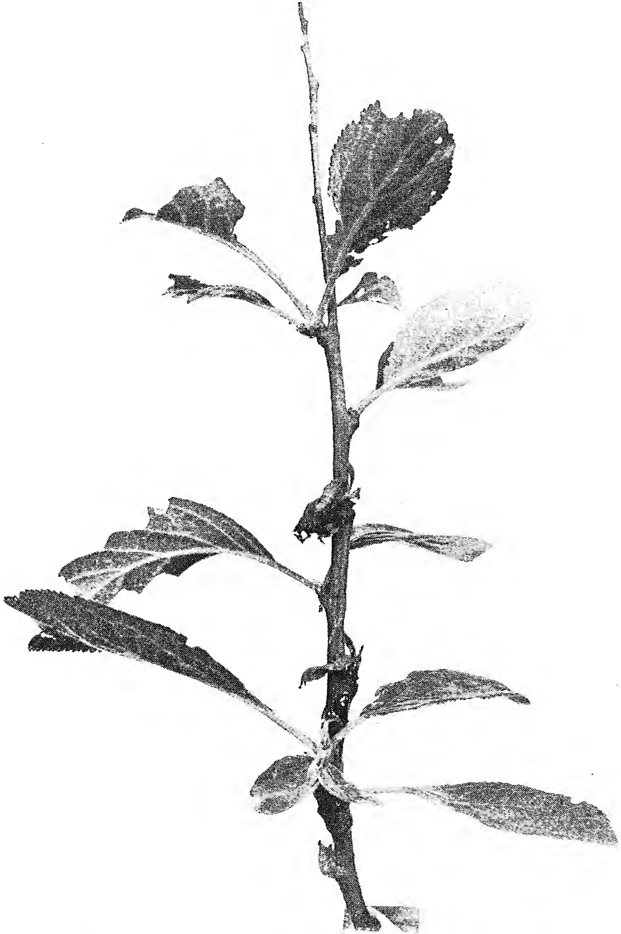
PLATE XIII

[*Photo: Annals of Applied Biology*



A. Wither Tip of Plums
Winter condition

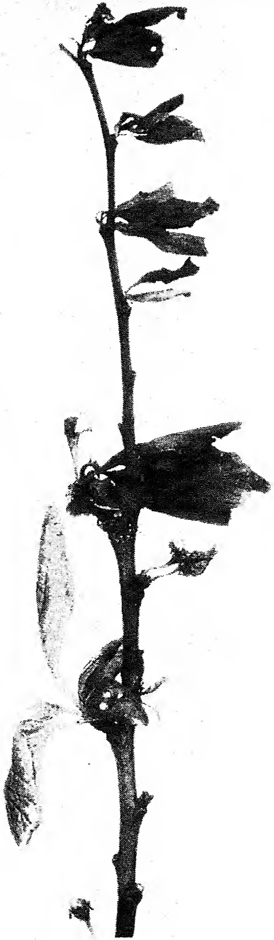
[Photo: Annals of Applied Biology



B. Plum Twig Showing Two Withered Short Shoots or Spurs

Infection probably came from the upper dead position which bears Monilia fructifications

PLATE XIV



A. Blossom Wilt of Plum
followed by Twig Blight

B. Plum Twig Showing A Dead Short Shoot
that should have developed into a flowering spur

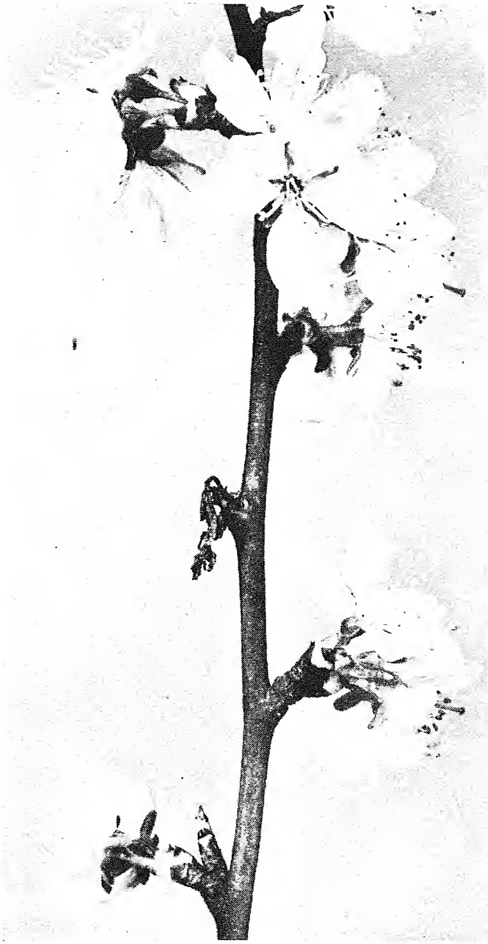
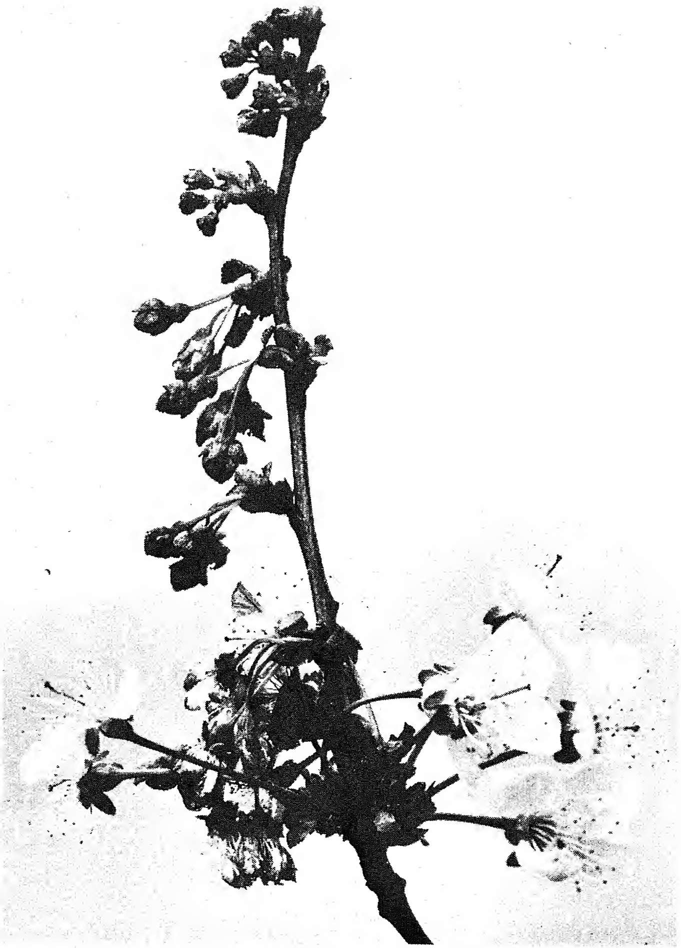


PLATE XV

[*Photo: Annals of Botany*



A. Morello Cherry
Blossom Wilt

followed by Twig Blight.
Infection through unopened
flowers

B. Morello Cherry
Blossom Wilt

followed by Twig Blight.
Infection through open
flowers

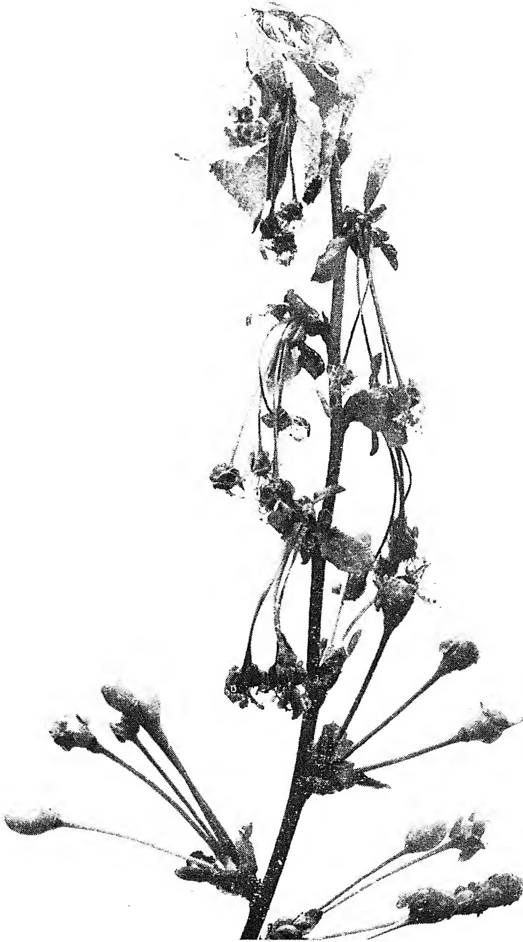
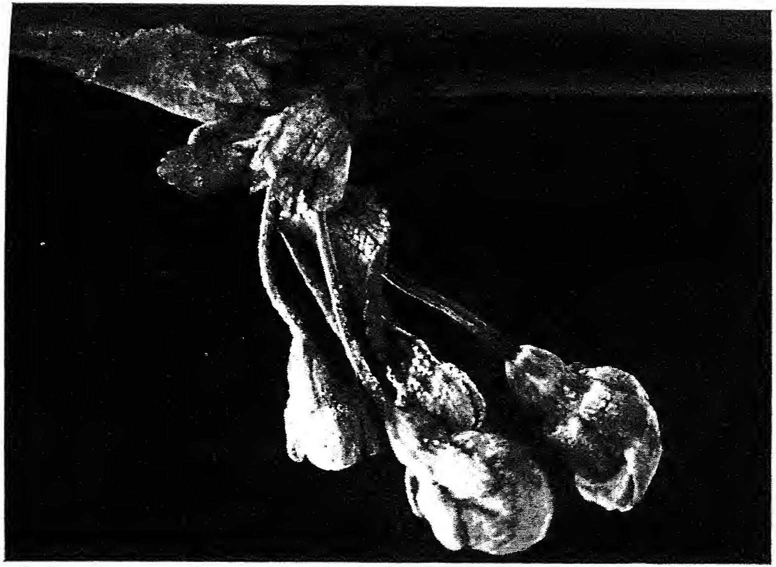
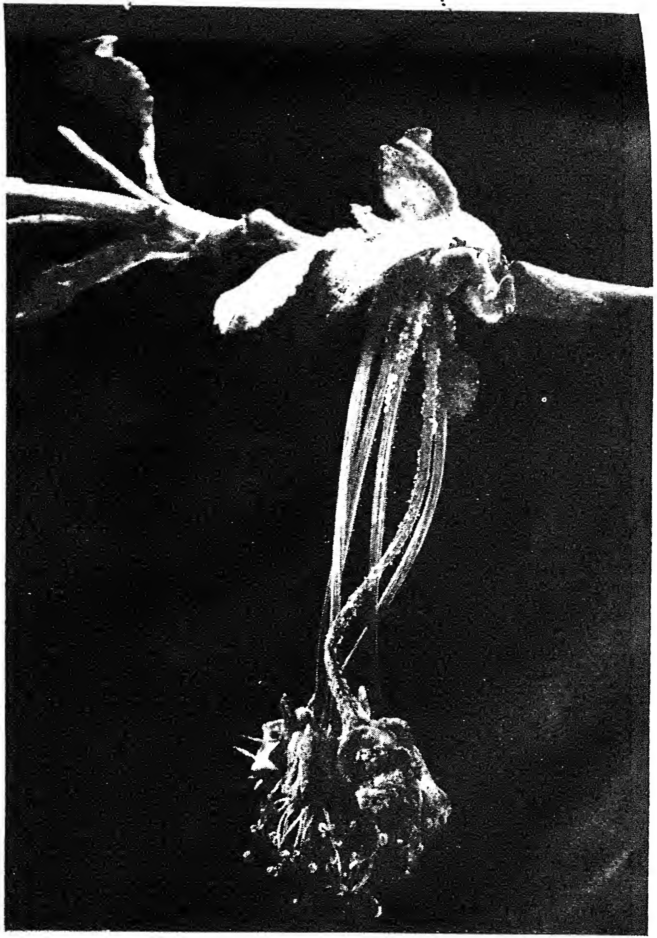


PLATE XVI

[Photos: J. Pomol. & Hort. Sci



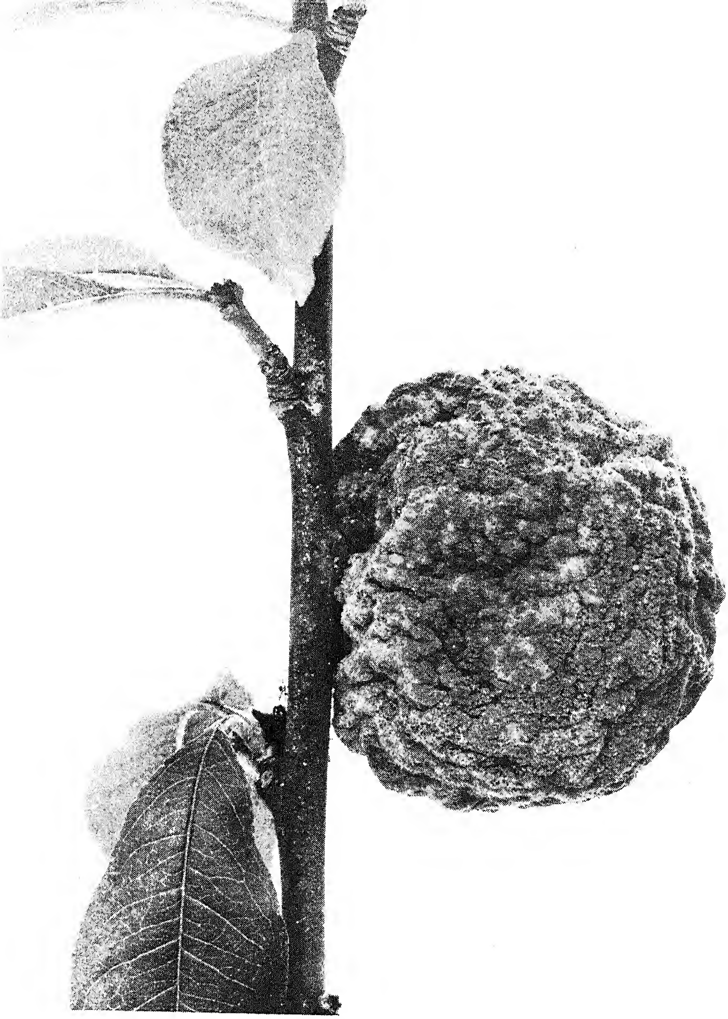
A. Infection of Unopened Morello Cherry Flowers
[× 2½]



B. Infection Through Open Morello Cherry Flowers
Infection started on the flower showing Monilia fructifications on the stalk

PLATE XVII

[*Photos: J. Pomol. & Hort. Sci.*



A. Peach infected with *Sclerotinia laxa*

B. Peach infected with *Sclerotinia fructigena*

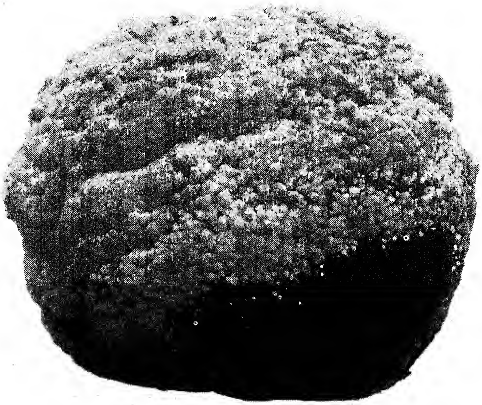
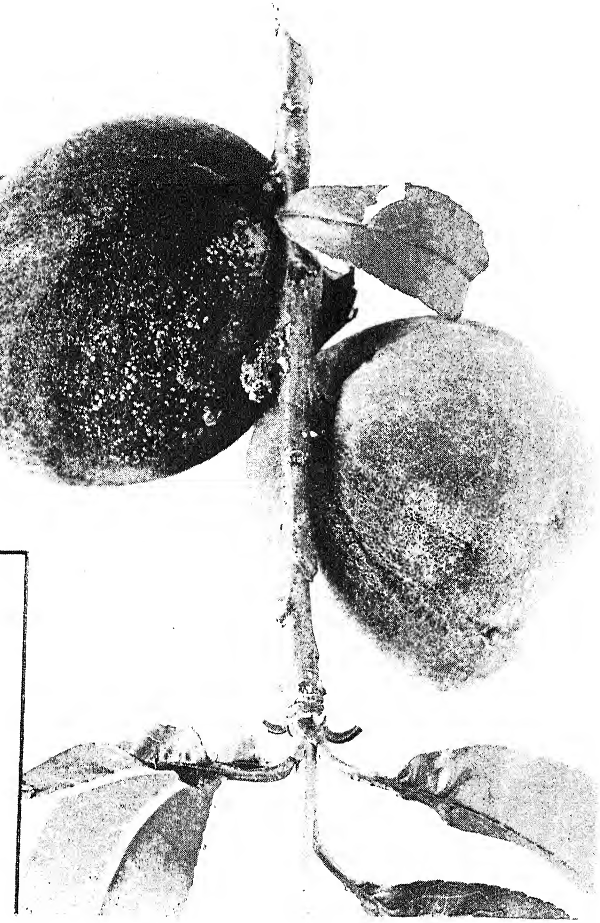


PLATE XVIII



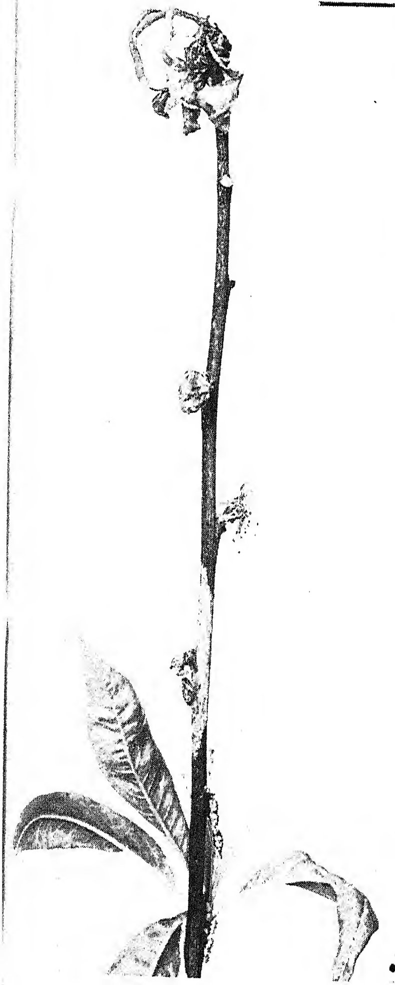
[*Photo: U.S. Dept. of Agriculture*

A. Brown Rot of Peach

(*Sclerotinia fructicola*)

The source of infection was the blighted blossom just below the rotted peach

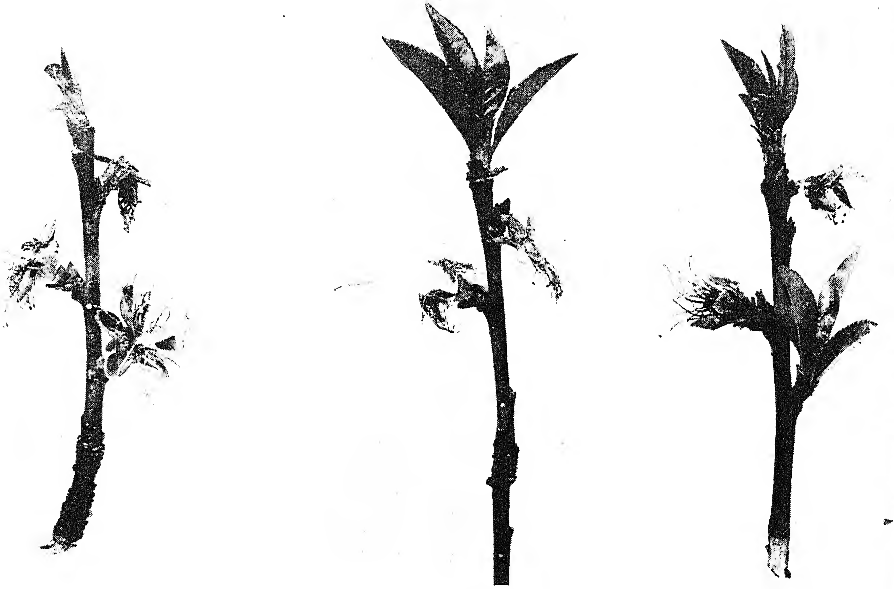
(From U.S.D.A. Tech. Bull. 328)



B. Twig Blight of Peach

following infection of a flower by *Sclerotinia laxa*. Note the pale cankered area around the infected flower

PLATE XIX

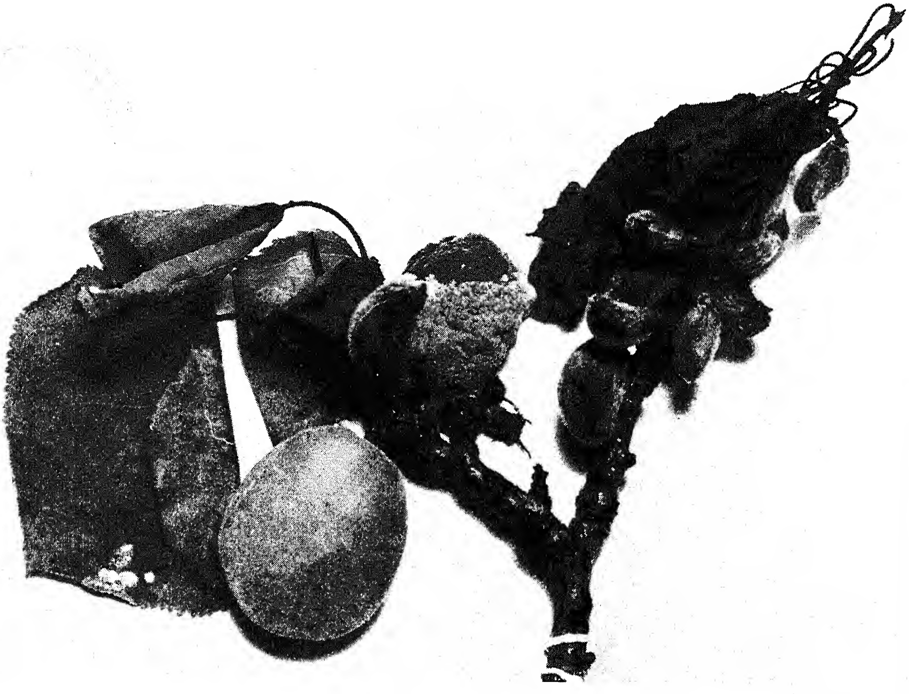


[*Photo: U.S. Dept. of Agriculture*

A. Blossom Blight of Peach (*S. fructicola*)

Note the stiff matted appearance of the floral parts of the infected (uppermost) blossoms

(From U.S.D.A. Tech. Bull. 328)



[*Photo: G. C. Wade*

B. Brown Rot of Apricot (*S. fructicola*)

PLATE XX

To supplement the record of *S. laxa* on *Prunus serrulata* the following information is given by Ingram (1948). With a few cherries, brown rot, or blossom wilt (*Sclerotinia laxa*) can be a tiresome and disfiguring disease, but happily the vast majority of varieties and species are not affected by it. It depends very largely on the type of inflorescence. Where the flowers are borne on short branches, even if these be killed outright by brown rot, no permanent damage will be done to the tree. If, on the other hand, the flowers are borne on short spurs arising from the main bough, the disease may cause serious injury to quite large branches, and sometimes these may even be killed by it. The Japanese varieties most likely to suffer in this way are Mikuruma-gaeshi and Okiku-zakura [varieties of *Prunus serrulata*]. Among the European cherries *Prunus cerasus* var. *rheseii* is the most susceptible. He advises planting the more susceptible varieties in open sunny situations, for the incidence of brown rot always seems to be associated with a still, humid atmosphere.

Other rosaceous plants have been mentioned either as natural hosts or as being infected from inoculations. E. F. Smith (1889) found it [? *Sclerotinia fructicola*] to grow in rose hips but not vigorously even under seemingly favourable conditions ; Reade (1908) collected "*Sclerotinia fructigena*" on the fruit of *Rubus occidentalis ;* Schellenberg (1923) mentions *Fragaria* as a host of the brown rot fungi. Harrison (1928) induced *Sclerotinia fructicola* to grow on loquat fruits (*Eriobotrya japonica*), while Sarejanni (1936) states that loquats in the Lamia district of Greece were very severely attacked by *Sclerotinia laxa* during a damp and rather severe cold spell in April, 1937, and Ling (1948) records *Eriobotrya japonica* as a host of *Sclerotinia laxa* in China.

Among host plants of families other than Rosaceae, the cultivated forms of hazel nut (*Corylus avellana*) appear to be very susceptible to infection by *Sclerotinia fructigena,* as observed in Austria (Wahl, 1924), Sweden (Eriksson, 1930, Johansen, 1937, Buchwald, 1943) and England (Wormald, 1944, Moore, 1950*a*), and Sorauer (1897) found that he could easily infect hazel nuts with the fungus.

Grapes (*Vitis vinifera*) are recorded as hosts of *Sclerotinia fructigena* in Czechoslovakia (Smolák, 1929), and in Georgia, U.S.S.R., by Nagorny and Issarlischwili (1929) and a *Vitis* sp. as a host of *Sclerotinia fructicola* in New South Wales (Biological Branch, N.S.W. Department of Agriculture, 1941*a*). Sorauer (1897) found that he could infect grapes with *S. fructigena,* and Schellenberg (1923) includes grapes among the hosts susceptible to infection by the non-specialized species of *Sclerotinia.*

Other plants mentioned as susceptible to brown rot infection are "Kürbis" (? pumpkin) and *Cornus mas* (Sorauer, 1897), currant and gooseberry (Ritzema Bos, 1904) and *Vaccinium* (Schellenberg, 1923). Harrison (1928) infected fruits of persimmon (*Diospyrus kaki*) and tomato with *Sclerotinia fructicola.*

That the brown rot fungi are able to infect so many species of plants is an important factor in relation to control measures. In efforts to reduce the enormous losses resulting from brown rot all possible sources of infection should be borne in mind, and hygienic measures such as removal and burning of all infected parts should be routine practice whenever possible. Such measures have frequently been emphasized with reference to fruit trees but it must be recognized that ornamental bushes, too, may become infected and the necessary steps should be taken to keep these also free from brown rot diseases, especially when, as in nurseries, ornamental bushes and fruit trees are grown in close proximity.

five days about half the surface will show the typical brown discoloration; meanwhile, blister-like swellings appear, usually in concentric rings around the point where the infection began and the fungus bursts through the skin to form buff-coloured, cushion-like tufts one-eighth of an inch across (Plate VIIB).

Infection may occur from soon after the fruit is set until it is stored, and also during storage. Although some injury is generally necessary for the fungus to enter, yet when an apple is infected the rot may extend from such a primarily infected fruit to any others in contact with it whether they are injured or not; thus, infected fruits are often seen in bunches (Plate VIIA).

Rotting apples may fall to the ground, where they continue to develop and shed spores, or they may gradually shrivel on the tree and become mummified. These mummies usually remain on the trees if they are not deliberately removed until the following year. The spores produced during the summer are blown or washed away, but in the following spring (May and June) new fructifications develop and a new crop of spores is produced to spread further infection.

BLACK APPLE. What is sometimes referred to as "Black Apple" or "Black Rot of apples" in this country, and as "Schwarzfäule" in Germany, is a form of brown rot that affects the fruit at, or near, maturity. It is occasionally noticed on fruit in the open, but it is more particularly a disease of stored apples, and is almost invariably present among apples during the first few weeks of storage; it has been observed, however, on apples that have been in cold storage for several months.

This type of rot starts as a brown rot but gradually the colour becomes darker; actual blackening starts as dark rings around the lenticels and eventually the whole skin becomes black. Fungus fructifications are few or absent on such apples, though frequently whitish tufts of mycelium appear on the surface. Since there is little or no rupturing of the skin, the black apples do not soon become shrunken as in typical brown rot, but remain for a considerable time with a firm shiny skin and of a consistency rather like rubber. Finally, if exposed to dry air, such fruits shrink and become grooved and indented; they remain quite hard and firm, however, and have an ebony-like black shiny surface. The whole fruit becomes a sclerotioid mass, the tissues being completely permeated by mycelium (Plate VIIC).

The absence of fungus fructifications on the black apples is probably partly due to storage conditions, for it was shown by Molz (1907) and confirmed by Hall (1933) that the Monilia fructifications are induced to develop by the action of light. Spinks (1916, 1917) found that some varieties, particularly cider apples, turn black more readily than others.

In Drummond's experiments (1934) young apples (inoculated in July) did not show any nigrescence, but mature apples from storage (inoculated in March) showed blackening in ten days. It would seem, therefore, that the stage of maturity at the time of infection is one factor determining the type of rot.

In Europe the black apple rot is almost invariably caused by *Monilia fructigena*. In one instance, however, isolations from a typically black apple yielded pure cultures of *M. cinerea*. When it was inoculated into apples, pears and damsons exposed to light the fruits became rapidly infected and grey sporodochia appeared within five days. Bramley's Seedling apples when inoculated and exposed to light produced a brown rot with numerous sporodochia while others kept in the dark, became quite black and bore only a few white barren hyphal tufts (Wormald, 1945). This effect of light and darkness is consonant with the results mentioned above for *M. fructigena*.

As this disease is merely a phase of brown rot in which the skin turns black under certain conditions, and in order to avoid confusion with the American Black Rot of apples, it seems preferable to refer to it as "Black Apple" rather than as "Black Rot."

The disease of apples generally known in America as "Black Rot" is caused by a totally different fungus, *Physalospora obtusa* (Schw.) Cooke, though *Sclerotinia fructicola*, the common brown rot fungus of North America, may also produce black apples similar to those infected with *S. fructigena*.

CORE ROT. Dowson (1925 ; 1926) described a core rot and premature fall of the fruit of certain varieties that ripen early and have soft mealy flesh, particularly White Transparent, but also, to a less degree, Domino, Langley Pippin and Duchess of Oldenburg. On most of the affected fruit there was a small brown patch about half an inch in diameter at one end. When cut open, these apples were found to be rotten at the core, and the same type of core rot was also seen in some apples that had no external sign of rot. Apples that were yellower and apparently riper than the rest while still on the tree were found to be affected with core rot. The pips of the affected apples were fully formed, but nearly all of them were soft and rotten. The dark brown, soft, rotten tissues contrasted sharply with the sound white parts.

CONTROL. It is evident that apple brown rot remains a serious and complex problem, and its various aspects still merit intensive study with a view to obtaining more satisfactory control of this disease.

In the early 1940's heavy losses from apple brown rot in Essex orchards led to observations on the underlying causes. The sources of infection were (1) mummified fruits overwintering on the trees, (2) infected fruits of the previous year's crop dropped and overwintered on the ground under the trees, (3) branches and fruiting spurs infected during the previous season and allowed to remain on the trees. Injuries, as courts of infection, caused by insect larvae, wasps and birds were noticeable. The growers were chiefly concerned with their losses in Cox's Orange Pippin, but infection was also severe on the varieties James Grieve and Laxton's Superb which served as pollinators for the Cox's, and also as sources of infection for that variety (Wormald & Moore, 1945).

General recommendations for the control of these fruit rots based on the observations mentioned above include:

The removal and destruction of all over-wintered sources of infection by early spring, e.g. mummified fruits, and infected spurs and branches, are essential operations.

Throwing all rotten fruits into the alley-ways, and not under the trees, is advisable so that they can be ploughed or disced in and thus rendered harmless.

Routine measures for the control of apple scab, sawfly and codling moth.

Wasps' nests in the neighbourhood of fruit trees should be sought out and destroyed.

Where birds are a nuisance by pecking the fruit some method of bird-scaring should be adopted.

Similar measures, where appropriate, for the control of brown rot should be applied to plum trees interplanted with the apples or growing near them, particularly with regard to the removal of mummified fruit after a bumper crop.

Fruits bruised and injured in any way, or picked without their stalks, should not be stored ; they probably carry spores from the orchard on their skins and infection will arise through the wounds at the stalk end or elsewhere.

Apples should not be stored when wet ; surface moisture should be allowed to evaporate before storage.

When the apples are growing in clusters, it is advisable to thin the fruit so as to obviate infection by contact with other fruit.

Co-operation in carrying out such measures among growers is necessary over as wide an area as possible, for the brown rot spores are very light and are carried in the air for long distances. *A neglected orchard is a menace to those around.*

If earwigs should prove to be troublesome by causing injuries allowing of infection, placing bands of sacking soaked with benzene hexachloride (BHC) round the trunks may be tried (see p. 33).

The above recommendations are, no doubt, a counsel of perfection but where losses have been serious over a series of years drastic measures are necessary.

With regard to Cox's Orange Pippin, in addition to these immediate measures, interplanting with resistant varieties, selected from present varieties or raised by breeding, should be considered.

To avoid rot developing on the fruit in storage, care must be taken during picking, sorting and packing not to damage the fruit. The apple pickers should be instructed to see that the apples are picked with their stalks : bruised and stalkless apples should be discarded for packing and storage.

Spraying for the control of brown rot of apples has not yet yielded very satisfactory results. Lime sulphur has been used for years without effecting any noticeable direct control and Bordeaux mixture has proved too harmful for apple trees (Moore, 1950*b*). Among the newer organic fungicides, Fermate showed some promise. Thus in trials by Bennett *et al.* (1945) cordon trees of Cox's Orange Pippin lightly infected with brown rot and heavily sprayed at the end of June with washes containing either copper oxychloride or ferric dimethyldithiocarbamate [ferbam] showed about 2 per cent fruit infected with brown rot in October as compared with 10 per cent in controls. The washes containing copper oxychloride caused serious defoliation and russetting, and the ferbam left a disfiguring deposit. Moreover the washes contained either arsenate or DDT and these may have contributed to a favourable effect against brown rot by reducing insect attacks. Marsh (1947) says that Fermate will not control a heavy brown rot infection of apples and the black deposit is undesirable.

Moore, (1950*b*) got no control with the organic fungicides used in his trials (ferric dimethyldithiocarbamate, tetramethylthiuramdisulphide and phenyl mercury chloride), and concluded that at least some of the new organic fungicides, including phenyl mercuric chloride, seemed unlikely to offer any more prospect than the older, inorganic ones, of controlling brown rot by protective spraying.

Winter applications of a phenyl mercury chloride preparation or of a calcium arsenite mixture was found to inhibit spore formation on mummified apples infected with *Sclerotinia fructigena* (Byrde, 1949). This offers a possible method of reducing brown rot of apples, but whether winter treatment of apple trees against *S. fructigena* is economic is questionable ; mummified apples are not so frequently seen as mummified plums in orchards, and, as bush trees are commonly grown now, the removal of mummies could easily be effected during the winter pruning operations.

There are several possible reasons for the lack of satisfactory control by spraying or dusting with fungicides in summer against apple fruit rot caused by *Sclerotinia fructigena*. It is probably partly due to the smoothness of the skin, so that a continuous layer of fungicide does not adhere, but chiefly perhaps because, in the apple, infection rarely occurs directly through the unbroken skin, and lenticel infection is uncommon. In peach fruit rot the fungicides are not so easily removed because of the hairs, and infection can occur through stomata and also down hair sockets (see p. 35); it may be prevented by applications of dusts or sprays, which are of little avail for the control of apple brown rot.

Canker

S. fructigena not only infects the fruit but may also invade the spurs and then the branches, and cause cankers. Infection of the branches by this fungus invariably occurs by way of the fruit. This becomes attacked first, and then the fungus extends along the fruit stalk into the spur and thence into the branch. When a branch becomes girdled the parts terminal to the canker die (Plate VIID). This type of canker has been observed on a number of varieties, including Melon (on which severe cankering, followed by the death of the leading branches has been observed), Cox's Orange Pippin, James Grieve, Cutler Grieve, Lord Derby, Stirling Castle and Lord Lambourne. When the cankers occur on young cordon trees, the stems may be girdled and killed above the cankers.

Monilia fructifications, similar to those which develop on the fruit, appear on the infected spurs and cankers; these fructifications lose their spores during the autumn and winter but new pustules arise in the following spring and summer, and another crop of spores is produced (Plate VIIIA). Such dead spurs and cankers, therefore, unless cut out, are like the mummified fruits, a source of infection.

These cankers can be distinguished from those described under Blossom Wilt, not only by the appearance of the fungus that develops on them, but also by the fact that infection occurs through the fruit and not through the flowers.

This type of canker can be prevented by removing the rotting fruit before infection has reached the spur. This operation may be practicable in a garden or a small orchard, but is probably impossible in large plantations, where attention must be given to the sanitary measures outlined above for brown rot of the fruit and all cankers must be cut out.

Blossom Wilt and Canker

Blossom wilt caused by *Sclerotinia laxa* f. *mali* varies considerably in intensity from year to year. In some seasons it is negligible in so far as actual damage is concerned. In others it may be so severe as to destroy almost the whole of the blossom on some trees. In fact, in some instances affected trees can be distinguished at a distance by reason of the masses of brown, withered flower trusses and leaves.

The disease was responsible for great damage in the south-east of England round about 1916. For some years after this comparatively little loss from blossom wilt was experienced, but in 1932 it again broke out with unusual intensity in Kent, Sussex and Cambridgeshire.

It occurs in apple growing areas of England and Scotland, and also in Eire (Boyle *et al.*, 1928). On the Continent it has been found in Sweden especially on the varieties Red and White Astrakan (Eriksson, 1913), in

Denmark (Buchwald, 1936), and probably also in Russia where a blossom wilt of apples is recorded (Lopatin, 1936) but attributed (probably erroneously) to *S. fructigena*.

Varieties very susceptible to this disease are Lord Derby, Cox's Orange Pippin, James Grieve and Ecklinville Seedling, but it occurs also on Duchess of Oldenburg, Worcester Pearmain, Allington Pippin, Early Julyan, Lane's Prince Albert, Lord Grosvenor, Prince Bismarck, Chelmsford Wonder, Newton Wonder, Bramley's Seedling, Domino, Beauty of Bath, Warner's King, Keswick Codlin, Duchess' Favourite, Rival, Fearn's Pippin, Dartmouth Crab, Ribston Pippin and Hanwell Souring. On the very susceptible varieties it is not uncommon to see from 50 to 70 per cent of the flowering spurs killed in certain seasons.

The most susceptible variety, Lord Derby, is grown on a considerable scale in the Weald of Kent, and in neglected orchards of this variety the disease has at times obtained such a hold that the trees have become unproductive and have had to be top-grafted with some less susceptible variety. Bramley's Seedling has been used for this purpose, for although not immune from this disease, it suffers but little from it.

Blossom wilt first becomes noticeable about a fortnight after the trees come into flower. Some of the trusses of flowers, with their surrounding leaves, begin to wilt and finally they wither and turn brown. Infection takes place through the open flower and about a fortnight after a single flower of a truss has become infected, the fungus has killed that flower and reached the tissues of the spur ; when this occurs the supply of sap is cut off from the other flowers and leaves of the cluster and they wilt in consequence (Plate IXA).

This wilting of the flowers is in itself often serious when many flower clusters are destroyed, but, in addition, the fungus frequently causes further destruction by extending into the branches to form cankers. When such a canker girdles a branch all the leaves and flower clusters on that part of the branch above the canker wither.

Trees of all ages, from the time they begin to bear flowers, are liable to infection. Large standard trees often suffer severely, but young trees are also attacked and sometimes the stems of cordon trees become girdled (Plate IXB).

During dry weather the fungus is not noticeable on the affected flowers but in wet weather (and also when the dead trusses are kept in a moist chamber) it appears at the surface of the flowers and flower stalks as grey conidial fructifications. If sections are made through the axis of a dead truss of flowers and examined microscopically, hyphae are invariably found and when particles of such sections are transferred to plates of a suitable nutrient culture medium, the fungus continues to grow and eventually produces its conidial fructifications.

The withered flowers and leaves remain attached to the spurs and may still be seen on the trees after the normal leaves fall in autumn. On the spurs and cankers the fungus does not come to the surface until the winter. During the winter and spring following infection, grey Monilia fructifications appear on the cankers and spurs and on the remains of withered flowers and leaves (Plate VIIIB). The conidia are dispersed in the spring and, falling on the flowers as they open, give rise to further infection. Thus, any cankers or dead spurs left on the trees until the blossoming period are potential sources of infection.

If a tree showing serious blossom wilt is carefully examined it will be found that where the wilted trusses are most crowded, there will be one or more old spurs (killed by blossom wilt infection the previous year) in the vicinity, usually on the same tree and a little distance above the newly infected flowers. Often the infection of ten or twelve trusses can be traced to a single dead spur left on the tree from the previous year, and under conditions particularly favourable for infection, that number may be considerably exceeded. Thus Plate XA shows 7 old dead spurs with spore pustules on them found on 10 trees, and 211 trusses of flower that became infected from those old spurs.

Growers have thought that this disease is brought about by frost but experiments have proved that it may be induced by placing conidia of the fungus on the stigmas of apple flowers. Some of these experiments were carried out in the open, others in a cool greenhouse where the action of frost as a factor was excluded. When a single flower of the truss was inoculated the progress of infection was usually as follows: the stigmas turned brown about the second or third day; on the sixth day the styles were brown to the base and two days later the stamens collapsed. The fungus then grew inside the flower stalk reaching the spur where it continued to grow and the other flowers and leaves on the spur began to wilt about the tenth day. In one experiment where the infected trusses were on short spurs (about half an inch long) borne on the main stem of a young tree, a sunken area appeared on the stem at the base of each spur in 16 days and the stem was completely girdled in 22 days.

The fungus causing blossom wilt of apple trees is *Sclerotinia laxa* f. *mali.* Morphologically it is indistinguishable from *S. laxa* as found on plum and cherry, but in inoculation experiments it has been found that *S. laxa*, taken from plums and cherries and inoculated into apple flowers, does not extend into the spur to cause the characteristic blossom wilt although it may kill the flowers actually inoculated. There is reason for believing therefore that attacked plum and cherry trees are not sources of infection for blossom wilt of apple trees.

The fact that the blossom wilt of apples is invariably caused by *S. laxa* f. *mali.* in Britain (so far as is known) and not by *S. fructigena* is of some theoretical and practical interest. Woronin (1900) records that he was able to induce it by inoculation with *S. fructigena* and Kotte (1948) mentions blossom infection as caused by this fungus. It would thus seem that *S. fructigena* is able to infect apple flowers but that its conidia are not usually in the air when apple flowers are open. In Britain fresh conidia of *S. fructigena* do not usually develop until towards the end of May or early June, while fertile sporodochia of *S. laxa* (including f. *mali*) may be found in winter and they produce conidia freely in spring. In this connection, Kotte remarks that the spores of *S. fructigena* mature later than those of *S. laxa* and generally blossoming is over when the spores of *S. fructigena* are dispersed, Occasionally, however, particularly when the blossoming period is protracted owing to rainy weather, apple flowers become infected, the fungus infecting the whole inflorescence and surrounding leaves.

CONTROL. The rational way of dealing with blossom wilt is to remove and burn all cankers and infected spurs. The best time to carry out this operation is immediately after wilting is observed in spring, for then the withered trusses of flowers and leaves are very conspicuous and, with care, few need be overlooked. Moreover, if this is done in time, the fungus may be prevented from extending into the branches. All affected wood and bark must be cut out, however, for if this is not done the fungus will continue to develop. If the labour necessary for carrying out this operation is not available at the time advised, the trees should be examined during the winter. Most of the cankers and dead spurs will be found without difficulty, even in winter, by reason of the remains of dead leaves and flowers on infected spurs but some are usually inconspicuous and may be overlooked unless special care is taken.

Attemps to control this disease by spraying have been made, but with little success. It has been found, however, that a wash consisting of caustic soda 10 lb., soft soap 10 lb. in 100 gal. of water, applied just before the buds begin to swell, helps to destroy the fungus pustules on cankers and spurs.

The recently tried eradicant sprays and those which have been used for spraying trees in blossom have not yet been tried against apple blossom wilt but appear to be worthy of trial.

PEAR

Fruit Rot

S. fructigena causes a brown rot of pears similar to that of apples, and the concentric rings of buff coloured Monilia fructifications on ripening pears are often a conspicuous feature in pear plantations (Plate XB). Infected pears may fall to the ground and the fungus continues to develop on them and shed spores for some time, or the fruit may remain on the trees and become mummified. As in apples, these mummies, if left on the trees, will develop a new crop of spores the following year and so give rise to further infection.

S. laxa has occasionally been found infecting young pear fruits ; on pear trees this fungus is not generally a serious parasite, but it has been found associated with blossom wilt.

Blossom Wilt

Blossom wilt (*Sclerotinia laxa*) of pears, in its general symptoms, is similar to that of apples except that it is confined almost solely to the spurs, and there is no extensive cankering on the branches (Plates XIA, XC). It appears to be comparatively rare in this country for it has been noted on only very few occasions.

It was first recorded for Britain in 1920 when it was found in Cambridgeshire. The following summer it was observed on Fertility pear trees at Wye, Kent ; on one tree there were 22 wilted trusses, about 4 per cent of the total number borne by the tree. All the flowers and leaves on each infected spur were dead, but the fungus had not extended further than the base of the spur, and therefore there were no cankers on the branches. Most of the dead spurs were pruned away during routine pruning operations, but some were overlooked and the disease appeared again the following year.

The disease was not noted again until 1925, when it was found in a plantation of Fertility pears at Wisborough Green, Sussex. In this instance it was estimated that there were from 70 to 80 dead trusses of flowers on each tree, the trees being 6–8 ft. high. Old dead spurs bearing Monilia pustules indicated that the disease had been present the previous year. The old fungal fructifications were present on the flowers, pedicels, petioles and the axis of the spurs ; on the newly infected trusses the pustules were on the calyx tubes and pedicels only. In 1941 blossom wilt was found on Conference pear trees at East Peckham, Kent ; fructifications of *Sclerotinia laxa* were found on the spurs and dead flower stalks early in 1941. thus indicating that blossom wilt had occurred in 1940.

This disease probably occurs occasionally on the Continent, as brief references to a similar type of infection on pear trees in Sweden and Italy have appeared. It is also found in North America, for Jackson (1915*a*) described a pear Canker in Oregon ; the fungus associated with the disease was given the name *Monilia oregonensis*, but it has since been shown that the fungus is indistinguishable from *Sclerotinia laxa* of Europe.

Pear blossom wilt can be brought about by inoculating flowers with conidia of *S. laxa.* Inoculation of the petals resulted in a brown discolouration but most of them dropped off before infection extended into the centre of the flower, and only in one instance did the flower, and finally the spur become infected.

Inoculation of the stigmas was more successful and the progress of infection could be followed. In these experiments, where from 2 to 5 flowers on each of a number of flower trusses were inoculated, the rate at which infection proceeded was rather slow at first, and about a week was required for the spores to germinate and the hyphae to grow down the styles into the centre of the flowers. Further progress was then more rapid and the fungus extended downward along the flower stalk towards the axis of the inflorescence at the rate of about one centimetre a day. In 11 days after inoculating the stigmas the fungus had reached the spurs, with the result that all the flowers and leaves on those spurs began to flag, and on the following day were completely withered.

The fungus causing blossom wilt of pears is indistinguishable in its morphology and cultural characters, from *Sclerotinia laxa* on plum and cherry trees, and it has been found that this fungus obtained from plum and cherry can infect pear flowers and cause blossom wilt. It would appear, then, that these three host plants when affected with this disease may serve as sources of infection for each other.

CONTROL. Since infection is brought about by the spores that develop on the spurs infected the previous year, control is best effected by the removal of all dead spurs. As in apple blossom wilt, this should be carried out as soon after flowering as possible, for then the withered trusses of leaves and flowers are easily detected.

Up to the present, this disease in Britain has been found only on the varieties Fertility and Conference. When these varieties are grown as bush trees, the cutting out of dead spurs should not be difficult. No very serious outbreaks have yet been reported and spraying as mentioned for apple blossom wilt is probably unnecessary. Plum and cherry trees in the vicinity of pear trees should be kept free from brown rot for, as mentioned above, *S. laxa* on those hosts can infect pear flowers.

QUINCE AND MEDLAR

On quinces, fruit rot caused by *S. fructigena* is common in Britain and on the Continent and the circles of Monilia pustules are often a conspicuous feature of the ripening fruit (Plate XIB). *S. laxa* is rare on the quince ; it was found on a young mummified fruit at East Malling in 1925.

Harrison (1928) reports that in Australia the quince is susceptible to brown rot infection by *S. fructicola,* particularly when damaged by codling moth larvae or by mechanical agencies, a loss of 20 per cent of the fruit being not uncommon.

S. fructicola is sometimes found on quinces in the United States. Fischer (1930) stated that brown rot was the most common and destructive of quince diseases found in Illinois markets during the previous autumn and winter.

Sclerotinia laxa has been observed on a mummified flower and on a leaf of medlar in Kent but such infection appears to be rare. *S. fructigena* has been found once on medlar fruits at West Malling, Kent.

Brown rot of medlars is thus of little importance in Britain and probably in most other countries. In Italy, however, Ferraris (1928*b*) reported a loss of 50 per cent locally, the fungus, *S. fructigena,* attacking the young fruits soon after the fall of the corolla ; the infected fruits became mummified and served as sources of infection the following spring. He recommended, in addition to hygienic measures, two applications of Bordeaux mixture or 3–4 per cent Caffaro paste in February, and after flowering two applications of 1 per cent Caffaro powder.

Leaf blotch of quince and of medlar are described on an earlier page (see p. 22).

PLUM

Fruit Rot

In Europe, brown rot of plums is caused by *S. fructigena* and *S. laxa* (Plate XII). It was for long considered that *S. fructigena* is almost confined to core fruits, but in recent years the loss due to fruit rot by *S. fructigena* in plums has been greater than that produced by *S. laxa.* Often the two fungi may be found on the same tree, sometimes on the same cluster of fruit or even side by side on the same plum.

Infection usually starts at a wound but when an infected fruit is in contact with a sound one a pad of mycelium is formed at the point of contact enabling the fungus to penetrate into the healthy plum and start a rot. In this way large clusters of plums often become infected, and it is not uncommon to see bunches of " mummies " hanging on plum trees through the winter (Plate XIIIA).

The disease may be very severe when conditions are such that mechanical or physiological injuries appear on the fruit. Insect punctures are the usual wounds that allow brown rot infection but in certain seasons other forms of injury are important in this connection, as in 1933, when hailstorms in June bruised the skin of the fruit, thus enabling infection to start, and brown rot continued to be severe, although the season was a dry one. In wet seasons, and particularly when a wet period succeeds drought, the skin of the fruit often splits, again allowing the brown rot fungi to enter.

In July, 1945, severe brown rot of the fruit of an Oregon Gage tree occurred in a garden at East Malling. On all the affected specimens examined (except one bruised) the rot was associated with a crack 3–5 mm. long at the style end. This variety appears to be subject to this kind of injury which renders it very susceptible to brown rot attack and a menace to other stone or pome fruits growing near.

Brown rot has been found to be the cause of serious wastage in plums in cold storage (Wormald & Painter, 1935, 1937). Under these conditions *S. fructigena* is responsible for most of the rotting but *S. laxa* may also be present. The infected plums often adhere in clusters, showing that, as in the orchard, one infected fruit may cause the rot to extend to others in contact with it.

On many of the rotting plums, infection was seen to have started at the stalk end when the fruit had been gathered without stalks. If cold storage of plums is adopted in order to extend the marketing period, care should be taken, particularly with the more valuable dessert varieties, to pick them with their stalks.

In Great Britain, no special control measures are directed against brown rot in plums except collecting and burning the mummified fruit, which is a very important measure and should be carried out more frequently and thoroughly than is often done.

In North America, Australia and New Zealand, brown rot of plums is caused by *S. fructicola*. Valleau (1915) found that infection may take place through the uninjured skin at any time during the development of the plum fruit, the hyphae entering through the stomata and lenticels. Varietal differences in susceptibility were found to depend on the structure of the lenticels. In wet seasons, *S. fructicola* is responsible for great losses and several sprayings are generally necessary, particularly as the fruit is often transported long distances by rail, and unless special precautions are taken, the disease spreads rapidly during transport. The methods adopted for controlling brown rot in plums caused by *S. fructicola* are similar to those described under peach brown rot (see p. 00).

Wither Tip and Spur Blight

The young leaves of plum trees are susceptible to infection by *S. laxa*. It has been found experimentally that infection may occur on the uninjured leaf (Drummond, 1934), but more readily through some injury and it is probable that wounds caused by insect larvae and aphids favour leaf infection.

When a leaf becomes infected, the fungus extends through the leaf stalk into the shoot and so produces shoot wilt. If the shoot is a long terminal one, a lesion appears round the base of the infected leaf, and when this lesion has girdled the shoot, that portion terminal to the infected area soon becomes flaccid and the leaves flag; this causes the soft green tip of the shoot to curve downward and the shoot wilts (Plate XIIIB). This curved wilted condition of terminal shoots is known as wither tip.

Later, the whole of that portion beyond the node at which the fungus entered becomes dark brown and withered and the discolouration also extends downwards below this node for a short distance, the lower limits of the lesion contrasting with the green healthy tissues. As further desiccation proceeds, the affected tissues harden and the withered leaves remain on the tree after the normal leaves have been shed. These dead twigs with their clinging dead leaves may easily be recognized during the winter and early spring (Plate XIVA).

If the infection starts on a leaf borne by a short shoot (spur), all the leaves of that spur soon wither and the fungus extends into the twig or branch bearing it with the result that a canker arises. Such cankers may girdle one-year-old twigs and cause the death of their terminal parts but often the development of the canker is arrested before this happens and callus develops around its margin. The infected tissues become disintegrated and gum often exudes in drops. A characteristic feature of these cankers when cut lengthwise is a dark line extending for some distance, (up to 4 in.), in the wood above and below the actual canker; this is caused by the disintegration of the young wood elements in those regions.

Usually the fungus is not visible on the shoots or cankers during the summer. It passes the winter in the infected tissues and appears at the surface during winter and spring as grey tufts typical of the Monilia stage of *S. laxa* (Plates XIVB, XVB). As the same fungus can infect the flowers and fruit, it is evident that these withered shoots and cankers are a source of infection resulting not only in further shoot wilt but in blossom wilt and brown rot of the fruit also, and they should not be allowed to remain on the trees through the winter. The smaller infected spurs and the cankers are not easily detected and the removal of withered tips will probably need to be supplemented by some spray treatment as mentioned below under Blossom Wilt and Twig Blight.

A form of wither tip has been seen in nursery layer rows and stool beds of plum stocks, the whole portion above ground of some of the shoots wilting completely. On uncovering the base of such a shoot a discoloured area is found and the tissues of the lesion contain the mycelium of *S. laxa*. Inoculation experiments have shown that this condition can be induced by inoculating the leaves of young stool shoots with conidia of *S. laxa* and covering them with soil as in the earthing-up that normally takes place in layer rows. It is recommended therefore that layer rows and stool beds of plum stocks should not be set out in the vicinity of plum trees infected with *S. laxa*.

Blossom Wilt and Twig Blight

In certain seasons blossom wilt (*Sclerotinia laxa*) of plum trees is a serious disease, rendering some trees almost barren. As in apple blossom wilt, the fungus extends from the flower primarily infected into the spur bearing it and sometimes into the young branches, thus producing cankers which cause the death of parts terminal to them. When many young branches are killed, the brown withered leaves and flowers give the trees a " blighted " appearance (Twig Blight) (Plate XVA).

The chief sources of blossom wilt infection are the mummified plums which are allowed to remain on the trees through the winter (Plate XIIIA). Serious blossom wilt may appear, however, when there are no mummies in the immediate neighbourhood ; in such cases it may be traced to cankers and dead twigs and spurs which had become infected the previous year and being left on the trees, had produced the fructifications that served to infect the flowers as they opened in the spring. In such instances, it will be found that the blossom wilt is most severe in the vicinity of the old dead spurs, twigs and cankers.

CONTROL. Mummified plums should be collected in winter and burnt, for they are all sources of infection for the new fruit, while those bearing *S. laxa* will produce spores which infect the flowers.

Twigs killed by blossom wilt should, whenever possible, be removed and burnt immediately they are observed in spring, for the dead flowers serve to disseminate the spores which may infect the fruit. Again, if the cutting out is done early, the diseased parts are more easily seen then than later. Moreover, in summer, the trees are less liable to become infected by the silver leaf fungus (*Stereum purpureum*) through the wounds necessarily made in cutting out, and this is another good reason why all necessary cutting out should be done during the summer months and not postponed until winter.

These sanitary measures, recommended for freeing the trees from sources of infection, should be carried out whenever possible as they are the most certain means of reducing losses due to blossom wilt. Some measure of success has been obtained by winter washing with one of the tar distillates or with a caustic wash (10 lb. soft soap and 10 lb. caustic soda in 100 gal. water). In field trials Lees and Briton-Jones (1925) found that a " Carbofluid " applied as a 7 per cent solution when the trees and inter-crops were dormant (end of January) completely controlled brown rot whilst the 4 per cent solution gave fairly good results. Spraying with Bordeaux mixture or lime sulphur immediately before the flowers open would probably also reduce the chances of infection, as in cherries (see p. 75), but at present there is no experimental evidence of the efficacy of these sprays on plums in Britain.

Mention should here be made of preliminary experiments carried out at the Long Ashton Research Station (Byrde, 1949, 1950) as suggesting possible measures for brown rot control of plums. Winter application of phenyl mercury chloride or of sodium or calcium arsenite preparations inhibited spore formation on mummified plums infected with *Sclerotinia fructigena.* Sodium dinitro-*ortho*-cresolate and sodium pentachlorphenate also showed some promise of possible winter treatments. In a field trial brown rot infection of plums of the current season was considerably reduced by a late June spray with phenyl mercury chloride (0·0035 per cent), Phygon "XL" (0·1 per cent) and copper 8-quinolinolate (0·1 per cent).

CHERRY

Fruit Rot

In Europe, fruit rot of cherries is caused by either of the two fungi *S. fructigena* and *S. laxa* as in plums; sometimes one fungus predominates, sometimes the other. On the young fruit, *S. laxa* is the more serious, for infection by this fungus may arise not only from spores produced on the mummified fruit, but from old dead spurs and also from flowers infected the same year, because in wet weather numerous spore pustules develop on the infected flowers. Later, *S. fructigena* may also take part in the infection, particularly if wet weather causes the fruit to crack. *S. fructigena* does not infect the flowers, so that primary infection of the fruit by this fungus occurs from conidia developing on mummified fruit.

Infection begins at some wound and a frequent cause of predisposition to rot in cherries is the splitting of the skin that takes place when wet weather is experienced just before or during picking. The fungus may pass from an infected cherry to a sound one in contact with it so that mummified cherries often hang in clusters.

Both fungi can infect acid cherries but brown rot of the fruit is far less serious in these varieties than in sweet cherries. On the Morello cherry *S. fructigena* has been found to infect the fruit, then, extending along the fruit stalk, it causes a lesion with exudation of gum on the twig.

CONTROL. Control measures in Britain consist chiefly in the destruction of mummified fruit and the removal of rotting fruit from the trees at picking time. A post-blossom application of Bordeaux mixture is recommended when blossom wilt has been prevalent for the fungus (*S. laxa*) that appears on the infected flowers infects the fruit.

In North America, New Zealand and Australia fruit rot of cherries is produced by *S. fructicola* and in these countries control measures are practised as on peaches (see p. 78).

Losses from the cracking of cherries with the subsequent fruit rot appear to be very serious in some parts of N. America particularly on Bing cherries (Gerhardt *et al.*, 1945), and experiments on the prevention of cracking in wet weather have been carried out not only in America but also in Europe. In tests by Powers and Bollen (1947) copper sulphate gave control of cracking in immersion tests with Bing cherries and preliminary field trials indicated that its use as a spray or dust will check cracking due to rain; the solution or spray to be effective and non-injurious should have a concentration of 0·1 to 0·01 per cent. Knoppien (1949) in the Netherlands records the percentage of cracked fruits of Meikers cherry, sprayed with 0·01 per cent of copper sulphate as 1·5 (untreated 5) and the percentage of fruits attacked by *Monilia* 4 (untreated 28·5); for the variety Varikse Zwarte, the corresponding figures were 2·5 (untreated 19) and 4·5 (untreated 10·5). Siaens (1951), using single applications of copper sulphate 0·01 per cent, Cuprosyl 0·01 and 0·5 per cent, and summer oil 1 per cent three or four weeks before fruit picking, obtained the best results with the Cuprosyl sprays. Whether or not such measures are economic has not yet been shown but they should be borne in mind by fruit-growers in regions where severe infection of cherries occurs year after year.

In this connection it may be noted that Palmiter and Hamilton (1943) observed that sweet cherries treated with sprays containing self-emulsifying cotton-seed oil had less cracking than those which were unsprayed and they attributed this to the oil forming over the fruit an invisible coating which did not allow absorption of rain water. This suggests that such an adjuvant to a spray fluid may assist in controlling brown rot by preventing cracking.

Wither Tip

This disease is comparatively rare on cherries. The effect is similar to that of wither tip in plums; leaf infection is followed by a lesion on the young shoot which wilts and curves over, eventually becoming desiccated. The infrequency of its occurrence makes it of little account from the point of view of actual damage but it should be noted that such shoots, if left on the trees, become a source of infection for flowers and fruit.

Blossom Wilt

Blossom wilt (*S. laxa*) of cherries is a common disease not only in Britain but on the Continent and also in the Pacific coastal regions of North America. It is caused by the same fungus that is responsible for blossom wilt in plums.

Infection probably usually occurs by spores falling on the stigmas for it has been found experimentally that infection readily occurs when conidia are placed on the stigmas of the open flowers.

In sweet cherries (where the flowers are borne on spurs on the two-year-old twigs or older branches), when a single flower of a cluster becomes infected, the fungus extends into the spur, killing all the flowers and leaves on that spur and little or no cankering occurs.

On acid cherries (particularly the Morello), the flowers of which are borne in clusters (umbels) directly on the one-year-old twigs, the effect is more serious for infection of a single flower results not only in the death of the whole cluster but extends into the twig and causes the death of all flowers and leaves terminal to the node at which infection occurred (Twig Blight).

That the acid cherries are particularly susceptible to blossom wilt and twig blight has been shown not only by observations in this country but also by experiments carried out by M. Schmidt (1937) in Germany.

In Morello cherries, infection takes place not only through the open flowers, but also through unopened ones, particularly if the weather is wet or misty shortly before the flowers open. Thus in some seasons there are two waves of infection in Morello cherries. If the flowers are attacked while still in bud, the effect is seen about the time the trees come into bloom, when certain twigs bearing wilted leaves and flowers will be seen. If such twigs are examined, it will be found that near the lower end of the dead portion, there is a cluster of unopened flowers bearing grey Monilia pustules, indicating that infection originated there (Plates XVIA, XVIIA). As the flowers open, they in turn become infected and the effect of such infection is observed

10 or 12 days later when the fruit is just setting, because it takes about that time for the fungus to infect the flowers and extend into the twigs (Plates XVIB, XVIIB). On neglected acid cherry trees, in seasons favourable for the fungus, infection may be so severe as to destroy almost all the blossom, for as shown above, not only are those clusters of flowers killed that become directly infected but so are all other flowers borne terminally to them on the same twigs. If the node at which infection occurs is low down, a length of twig of twelve inches or even more, bearing 8 or 10 clusters of flowers, withers. When there are many such twigs, the trees have the appearance of having been scorched by fire.

During the summer, the spores which develop on the infected flowers become washed or blown away. The fungus, however, passes the winter in the tissues of the withered flowers, the dead spurs and the twigs and on these, early in the following year, Monilia fructifications develop and the conidia they produce infect the flowers in spring.

CONTROL. It follows that in order to reduce blossom infection to a minimum, all dead parts should be cut out and burnt. On Morellos (usually grown as bush trees), this is not an impossible operation but on large standard sweet cherries the removal of innumerable dead spurs is hardly practicable and spraying must be relied upon.

On acid cherries grown as bush trees, the trees should be examined at the time of flowering and again about a fortnight or three weeks later. All wilting twigs should be removed, for not only are such twigs sources of infection, which should be cut out in any case as soon as possible, but also if the operation is carried out at that time, it is likely to be more thorough than if left until later, for the twigs bearing wilted leaves and flowers are very conspicuous and cannot easily be overlooked. If they are allowed to remain on the trees until after the fruit is picked they will be partly hidden by the new growth, while in winter, although the larger dead twigs are easily recognized by their persisting dead leaves, the smaller dead spurs are not readily seen.

Salmon (1907) found that blossom wilt of Morellos could be prevented by spraying with Bordeaux mixture just before the trees came into flower and again immediately after petal-fall. The first application must have been effective against blossom infection since infection takes place through the flowers and the second against fruit infection. Lime sulphur applied immediately before the trees come into flower has also been found to check blossom infection in sweet cherries.

In Australia, Hammond (1922) advises that cherry trees should receive their first spraying when 10 per cent of the blossoms are open. Spraying as the trees are coming into bloom, using lime sulphur (1 in 30) has been tried in north Kent and good control has been reported. The petals of flowers actually open at the time may become scorched by such a spray, but the treatment seems to offer more certain protection to the flowers which open later.

Moore *et al* (1939) report that excellent control of brown rot blossom wilt of Morello cherries results from cutting out infected parts in spring and giving one application of tar oil (10 per cent) later in January and one of Bordeaux mixture (6:9:100) just before blossoming.

Thus for the control of brown rot in cherries, it is recommended that when practicable, all dead twigs, spurs and mummified fruits should be removed. For protection against blossom wilt the trees should be sprayed with Bordeaux mixture (6 : 6 : 100, increasing the lime by one half if hydrated lime is used) or lime sulphur (1 in 30 to 1 in 50) just before the flowers open or even when some of the flowers are already open. To protect the fruit, the trees should receive another application immediately the fruit has set, reducing the strength of the lime sulphur to 1 in 100 ; one or more applications may be necessary later if fruit rot threatens to be serious. These later applications are not usually necessary in Britain but are generally recommended where *S. fructicola* abounds.

PEACH AND NECTARINE

In Britain, brown rot is not a serious menace to peaches and nectarines because they are grown in such comparatively small numbers, chiefly under glass, that they can receive constant and careful attention. They are however often grown in the open on walls and peaches sometimes as bush trees. The chief risk in such cases is that they may become infected from plum and cherry trees in the neighbourhood.

Fruit rot and blossom wilt of peaches both occur in Britain but without disastrous results. In other countries however, where peach trees are cultivated on a commercial scale and the fruit often transported over long distances, brown rot of the fruit is a most disturbing factor of peach cultivation and special precautions must be taken to prevent its spread in the orchard, in storage and during transport. On the trees themselves, not only may there be serious loss from infection of the fruit but the flowers also become infected and blossom blight destroys not only flowers but kills twigs, and cankers are produced on larger branches by the fungus invading them from the flowers.

In the United States and in New Zealand a leaf infection has been described that results in brown spots which may eventually become excised and drop out. Roberts and Dunegan (1932) say that leaf infection is common but unimportant but that in moist weather the fungus may enter leaves injured by other agencies, especially by the leaf curl fungus, *Taphrina deformans,* and that when leaves are infected early they provide an abundance of conidia which may infect the ripening fruit. The association of leaf curl and brown rot is also mentioned by Steenland (1949) who considers that leaves on which he found sporodochia of *Sclerotinia laxa* were first infected by *Taphrina deformans* and that such a source of inoculum may, when present, play an important part in blossom blight infection the next spring if favourable weather for this disease occurs.

Fruit Rot

In Britain and in Europe, brown rot of peaches and nectarines is caused by both *Sclerotinia fructigena* and *S. laxa;* in North America, Australia and New Zealand by *S. fructicola* (*see* Plates XVIII, XIXa).

As in plums, the fruit rot begins as a small brown spot which rapidly increases in size until the whole fruit becomes involved ; meanwhile, numerous Monilia fructifications appear, eventually almost covering the whole surface, and myriads of conidia are disseminated to set up further infection. In America, this disease is sometimes known as "the rot" (Roberts & Dunegan, 1932), because in the United States, with the occasional exception of Rhizopus rot, it is the only rot of the peach that is of great importance to producer, shipper, carrier, commission merchant, retailer and consumer. All these at times suffer heavy financial losses because of brown rot.

The peach may become infected by *S. fructicola* without previous injury, through the stomata or hair sockets (Curtis, 1928), but in the orchard infection usually follows insect punctures. In the United States, such punctures are mostly caused by the plum curculio (*Conotrachelus nenuphar* (Hbst.)). The incubation period of the fungus is very short; in favourable circumstances the development of conidia has been observed to take place within twenty-four hours of inoculation (Quaintance, 1900).

The infected fruits may fall to the ground but many stay on the trees as mummies. The fungus passes the winter within their tissues and renews the production of Monilia fructifications the following spring.

The Sclerotinia stage of *Sclerotinia laxa* has not been found on mummified peaches, but that of *S. fructicola* is of very frequent occurrence (Plate Vc) and in Canada, the United States, Australia and New Zealand, the dissemination of ascospores in the spring is a factor of such importance that methods of controlling brown rot includes measures for the destruction of fallen mummified fruit.

Blossom Wilt

In Europe, peach blossom wilt is induced by *S. laxa*. This fungus infects the flowers probably mostly by way of the stigma, as in plums; it then invades the flower bearing twigs, causing these to wither (Twig Blight) (Plate XIXB).

S. fructicola produces a similar disease in those regions where it occurs (North America, Australia and New Zealand) resulting in blossom wilt (blossom blight), twig blight and cankers (Plate XXA). Roberts and Dunegan (1926) in their inoculation experiments with this fungus, found that all parts of the open blossoms the stigmas, anthers, petals and inner surface of the sepals may be attacked.

Both fungi pass the winter within the tissues of the dead flowers. mummified fruits, the twigs and the cankers, come to the surface the following year and produce and shed conidia. Where *S. fructicola* abounds, the flowers are liable to infection not only by these conidia but also by the ascospores liberated from the Sclerotinia fructifications on the fallen mummies.

McCubbin (1918), from a study of peach canker in Canada considered that the brown rot fungus acts as an initial agent only, and that while it may be responsible for the first stage of cankers, the yearly extension of these is caused by *Valsa leucostoma* which becomes established in the brown rot lesions. This was confirmed by Willison (1931–1935) who made a special study of peach canker in relation to the invasion of fungus wound parasites and by Willison and Chamberlain (1935) who found that common sites of infection by *Valsa cincta* are the twig and spur lesions caused by the brown rot fungus. The summer treatment of the brown rot fungus will thus help to control peach canker. Moore (1952) records an unusual instance of *Monilia fructigena* causing lesions on vigorous current year shoots of Peregrine peach; these lesions are smooth and brown, and at first small and circular, but later they elongate rapidly along the shoot and are often accompanied by copious external gumming. Infection appeared to start at lenticels. In a moist chamber the fungus was able to infect peach leaves. It was also shown experimentally that infection could start at union cuts made during budding operations when there were rotting peaches bearing *M. fructigena* in the vicinity. This kind of infection has not yet been seen to arise after normal budding in the nursery, but it does emphasize the necessity of destroying infective material.

CONTROL. When peaches and nectarines are grown in gardens and greenhouses, as commonly practised in Britain, control of brown rot can be effected by the prompt removal and burning of all rotting fruit, coupled with the cutting out of dead twigs. If this is carefully carried out, spraying should not be necessary. It should be borne in mind however that chance infection may come from other host plants in the vicinity, viz: *Sclerotinia laxa* from plum, cherry and certain ornamental shrubs, and *S. fructigena* from plum, cherry, apple, pear, quince, almond and possibly cobnut.

In some regions of the United States wild rosaceous shrubs are of frequent occurrence and they often become infected with *Sclerotinia fructicola.* Roberts and Dunegan (1932) found that many species of wild plums show all the different phases of the disease described for peach (blossom blight, twig canker, fruit rot, and mummy formation) and they have observed apothecia produced in abundance under trees in thickets. Wild plums may thus serve as fertile sources of infectious material. It is obvious that, when practicable, such sources of infection should be eliminated.

The accumulated information relating to the control of peach brown rot (*S. fructicola*) in the United States, where the disease is very destructive and has been investigated by many workers, was reviewed by Roberts and Dunegan (1932). The control of the disease depends on sanitary measures (removal of diseased fruit, the ploughing under of mummies and the cutting out of all cankers and infected twigs) and upon an adequate and timely use of fungicides, coupled with proper measures for the control of the plum curculio. These authors recommend a full spray or dust schedule for general use against brown rot of the fruit, based on their experiments and those of other workers. Many spray programmes have been recommended for peach-growing areas and growers are advised that their selection should be based on the recommendations of the plant pathologists who have carried out trials in their particular region.

Berkeley (1927) in Canada found a pre-blossom spray was very efficacious in controlling blossom blight; he used " wettable sulphur " when the blossom buds were showing pink. Fish (1927) in Australia, and Curtis (1929) in New Zealand, consider that two pre-blossom applications are necessary. Fish recommends lime sulphur 1 in 9 for the first application (just before the buds begin to swell), and for the second 1 in 35 (as the buds show pink), while Curtis used Bordeaux mixture 5:4:50 at bud movement, and lime sulphur 1: 50 at the pink-bud stage.

The " self-boiled " lime sulphur was recommended by Scott and Ayres (1910) as being less dangerous than either Bordeaux mixture or commercial lime sulphur when used on peach foliage. In its preparation the heat from the slaking of the lime causes the sulphur and milk of lime to become intimately mixed and form a suspension. Various substitutes for self-boiled lime sulphur such as colloidal sulphur, wettable sulphur and various forms of sulphur dust have become popular in recent years.

A spray mixture, zinc lime, prepared with zinc sulphate and hydrated lime has been tried on peaches by Roberts and Pierce (1932) who state there are indications that it will, at least, control mild cases of peach brown rot without injury to the trees.

Field trials with some of the new synthetic organic fungicides have been carried out but these preparations have not yet been brought into general practice against peach brown rot. Further work in this direction may yield interesting results.

In America and Australasia where *S. fructicola* abounds and produces apothecia from fallen mummies, the dissemination of ascospores should be prevented (see p. 55).

APRICOT

The apricot is particularly susceptible to brown rot infection as shown by numerous papers published in Europe, America and Australasia where severe losses have been reported, not only from infection of the fruit but also from infection of the flowers which leads to the invasion by the fungus of the flowering branches so that large parts of the affected trees are destroyed. The blossom blight phase under Californian conditions is fully described by Rudolph (1925) together with an account of field trials for its control. He suggests that the Japanese quince *Chaenomeles japonica* which is very susceptible to blossom blight, may serve as a source of infection for apricots.

In Britain the apricot is rarely cultivated commercially but it is sometimes grown in gardens though seldom in plantations. One paper on the control of brown rot in a garden has appeared (Denham & Wormald, 1942). Later Denham (1946) wrote that in his garden, infection of ripening fruit was regarded as more serious than blossom wilt and appeared to be caused by spores from plums in the vicinity. Salmon and Ware (1931) reported the occurrence of shield-shaped cankers caused by *S. laxa* on apricot branches.

Fruit Rot

As in other stone fruits, brown rot of the fruit may be produced by *S. fructigena, S. laxa* or *S. fructicola* (Plate XXb). As the damage caused by *S. fructigena* is primarily confined to the fruit, this fungus is of minor importance compared with the other two species. The fruit rot caused by *S. laxa* and *S. fructicola* is associated with blossom wilt, hence common measures of control are directed against both phases of the diseases.

In Germany Maier (1942*a*) observed apricots suffering from a destructive dieback of branches caused by *S. laxa* and *S. fructigena.* In many cases, the entire crown of a tree was affected and had to be removed. Infection was found to originate in the fruits and the mycelium of the fungi proceeded through the pedicels into the branches where it spread through the phloem and xylem, obstructing the supply of water to the leaves by the accumulation of hyphae, formation of tyloses and the deposition of gum. He also described severe infection of the fruit through splits and cracks (Maier, 1942*b*).

Blossom Wilt

In Europe, blossom wilt is caused by *S. laxa* and is sometimes very destructive. Severe losses have been reported in Switzerland, France (particularly the Rhône valley), Italy and in Russia in the regions bordering on the Black Sea. In the Crimea, where the cultivation of apricots used to flourish, blossom wilt and the subsequent cankering and killing back of branches has almost destroyed the industry. The intensity of the attack is enhanced by the proximity of the plantations to the sea and the frequency of sea mists during the blossoming period (Strelin, 1926 ; Balakhonoff, 1930).

The outbreaks in the Rhône valley have been investigated by a number of workers in Switzerland and France, particularly by Faes and Staehelin (1924), Chabrolin (1924) and more recently by Jöessel (1932*a*) who finds that infection takes place not only through the open flower by way of the stigma or through the inner surface of the calyx tube but also through unopened flowers. Chabrolin gives diagrams, drawn from serial sections, to illustrate the invasion of the stamens and ovary by the hyphae after initial infection in the calyx tube (Fig. 7).

The infected flowers wither and the fungus readily passes along the short pedicel into the twig so that the terminal portion of the twig and its flowers wither. The fungus passes the winter in the withered flowers and within the twigs, appearing the following year at the surface to cause further infection.

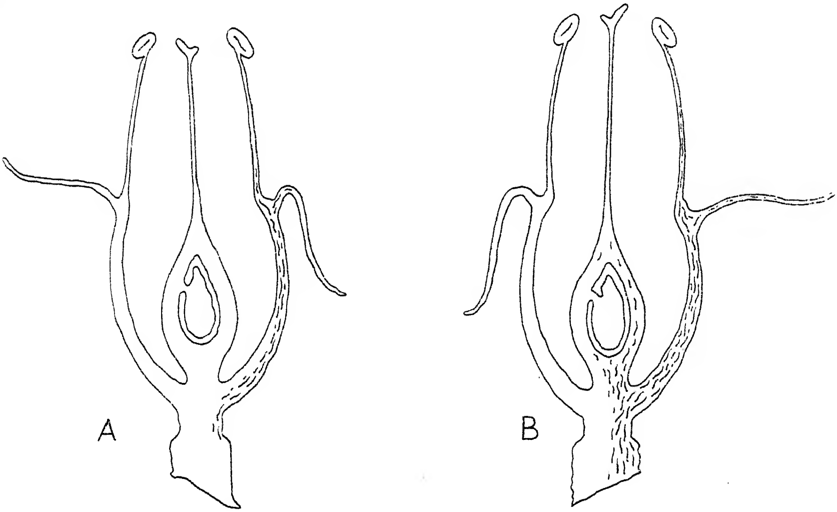


FIG. 7. Diagrammatic Section of an Apricot Flower

A. Partly invaded by *Sclerotinia laxa* showing (by broken lines), on the right, tissues containing mycelium in the calyx tube.

B. The mycelium has extended further, and has already invaded a part of the ovary and of the pedicel.

(from Chabrolin, 1924)

Jöessel (1932*a*) as a result of numerous experiments and observations recommends spraying twice with Bordeaux mixture, the first application as the buds are swelling and the second as the petals show between the sepals but before they expand.

In America, the apricot is grown in a number of States, but particularly in California, where its brown rot diseases are troublesome and have been investigated, particularly from the point of view of control, by Howard and Horne (1921) and later by Rudolf (1925).

Both *S. laxa* and *S. fructicola* infect the apricot in California. It is probable that *S. laxa* is chiefly responsible for blossom wilt and twig blight, for Barss (1923) found that this fungus (referred to at the time as *S. oregonensis*) is the cause of severe blossom infection of all kinds of stone fruits along the Pacific coast.

One reason for the increased virulence of apricot brown rot in California is, according to Rudolph (1925), that the resistant varieties Moorpark and Hemskirk are largely replaced by the highly susceptible Blenheim.

Yarwood (1948) states that apricot jacket rot, also called green rot, calyx rot and blossom rot, is caused by *Sclerotinia sclerotiorum, Botrytis cinerea, Monilia laxa* and *M. fructicola*, the relative importance of the fungi probably being in that order. Fruit rot by the three genera can be distinguished by the colour of the rot (being darkest with *Monilia* and palest with *Sclerotinia*).

Rudolph (1925) came to the conclusion that spraying with Bordeaux mixture was the best means of controlling brown rot in apricots. In badly diseased orchards situated in localities which are favourable to the ready development of the disease multiple spraying throughout the entire susceptible period was the only means likely to secure practical control but having brought the disease within the limits of practical control by the multiple spray method, fewer sprays, and in most instances, only one spray of Bordeaux 8:8:50 or weaker strength will be needed in succeeding years to hold the disease in check.

Although sulphur preparations are extensively used in America against peach brown rot caused by *S. fructicola,* they have been found to cause definite injury if applied to apricots and Bordeaux mixture is generally recommended for the control of apricot brown rot. The damage by sulphur preparations, sometimes referred to as "sulphur poisoning" is mentioned not only in America (Rudolph, 1925 ; Zeller *et al.*, 1942) but also in Australia (Anon., 1945 ; Hutton, 1947*b*) and New Zealand (Talbot, 1949).

Wade (1949) in Tasmania, recommends Bordeaux mixture 6:4:40 applied at the pink bud stage and warns that the trees must not be sprayed at any stage while leaves and fruit are on the trees because no material is known at present which can safely be used during that period. More recently (1951) in experiments with cover sprays, he reports that some of the newer fungicides give moderate control. Two of these, Isothan and Tetroc cause injury to leaves while Fermate leaves a conspicuous black deposit on the fruit. His results indicate however that a good measure of control of brown rot can be expected from the use of Thiotox (tetramethylthiuramdisulphide) which does not leave an objectionable residue and no adverse effect on the canned products has been detected.

Yarwood (1948) says that the twig infection phase of brown rot caused by *Monilinia laxa* is controlled by an application of 0·2 per cent Fermate with a spreader at full bloom.

CONTROL. From a general review of the observations mentioned above, the following recommendations may be given for the control of brown rot diseases of apricot:

A tar oil spray in winter, as late as possible but before the buds begin to swell, or an "eradicant" spray as mentioned above (p. 55) may be tried. As these measures have not yet been thoroughly tested on apricot, they should be employed with caution.

Spraying, just before the flowers open, with Bordeaux mixture 6:6:100 (or 6:9:100 if hydrated lime is used).

Cutting out as many infected twigs and spurs as possible, preferably in spring about a fortnight or three weeks after the trees come into bloom, for the infected twigs with their dead or withering flowers and leaves are more conspicuous then than they will be if left on the trees until the winter.

In countries where *S. fructicola* is the apricot brown rot fungus, it may be necessary to take steps to prevent the dissemination of ascospores (see p. 55).

ALMOND

Brown rot infection has caused serious damage to almonds in Europe (Greece and Crete) and N. Africa where it is caused by *Sclerotinia laxa,* and in the United States, particularly in California, where it is attributed to *S. fructicola.* In Crete, *S. laxa* is stated to reduce the crop by nine-tenths (Sarejanni, 1935).

In California, only one important commercial variety, the Drake, is usually severely affected by brown rot (Wood, 1937).

Flowers, leaves, branches and fruit are attacked. Primary infection is often through the flowers and then the fungus extends into the spurs and branches. Infection on the branches is said to start sometimes at small cracks (Bureau de la Défense des Végétaux, Maroc, 1941) in the bark but this appears to be exceptional. Canker may encircle the branches and then the terminal parts are killed.

Details of infection are given by Sarejanni (1935) for Cretan almond orchards, where infection falls into three stages. In the first, infection is mainly through the flowers, whence the fungus proceeds into the peduncle and the adjacent young branch. In the second stage the fruits become atrophied, and in the third the current year's branches are attacked. The last stage occurs at the end of April or early in May, when warm weather arrests further progress.

Preliminary experiments showed that fungicidal treatment is of no value unless preceded by severe pruning and careful orchard sanitation, including the removal of all cankers and mummified almonds.

Reference is made on an earlier page to the infection of almond fruits by *S. fructigena* on bushes grown in gardens as ornamental shrubs.

CONTROL. In Morocco the recommendations for controlling brown rot of almonds include the application of Bordeaux mixture, lime sulphur or colloidal sulphur just before the buds begin to swell ; 1 per cent copper oxychloride or 0·25 per cent colloidal sulphur as the trees come into leaf (Bordeaux mixture and lime sulphur are considered to be unsafe at this stage) ; and 1 per cent copper oxychloride or 2 per cent self-boiled lime sulphur after the calyx has fallen.

In California, Wood (1937) advises the application of Bordeaux mixture 8:8:50 at the " pink-bud " stage, and adds, that if rains occur between this stage and the end of bloom a second spray is necessary applied as soon as all the blossoms have emerged from the winter buds.

HAZEL NUT

A disease of cultivated hazel nuts (cobnuts and filberts), referred to in England as Nut Drop because the most obvious symptom is the premature fall of the nuts, is associated with the presence of the hyphae of *S. fructigena* within the affected nuts and the appearance of the Monilia fructifications on the external surface of the shells.

The first notice of this disease appears to be that of Sorauer (1899) who had observed it towards the end of June, 1887, in the arboretum of the Proskau Pomological Institute. He showed by experiments that the fungus from hazel nuts could be successfully inoculated into apples, plums, fruits of hawthorn, and grapes, and *Monilia* from apple into hazel nuts.

An illustrated description of the disease is given by Eriksson (1930). It has been found also in Austria (Wahl, 1924). Johansen *et al.* (1937) and Buchwald (1943) showed that the fungus was *Sclerotinia fructigena.*

It was first observed in England about the middle of July, 1943, when severe nut drop occurred on a fruit farm in Kent (Wormald, 1944). The fungus was identified as *Monilia fructigena ;* it was successfully inoculated into apples and plums on which typical brown rot appeared with *M. fructigena* fructifications. The disease in Kent has been further investigated by Moore (1947 ; 1950*a*) who finds that the fungus is generally a wound parasite following oviposition punctures by the nut weevil, *Balaninus* *nucum,* in the soft-shelled nuts early in July. The association of the nut weevil with infection by *Monilia fructigena* has also been observed by Hansen and Weber (1950).

In some cases however very young nuts become infected and the rot then spreads to others of the same cluster so that the whole bunch drops. This condition was observed on the Kent specimens but it had previously been described by Sorauer who suggested that infection in such instances arose from infection of the stigmas.

CONTROL. Protective fungicides have not proved effective against nut drop. To prevent fungal infection sources of *Monilia fructigena* (e.g. mummified apples and plums) on neighbouring fruit trees should be removed and destroyed during the winter. The application of sprays containing lead arsenate resulted in some reduction in rotting by controlling the weevil (Moore, 1950 *a*). It appears therefore that control measures should involve insecticidal, rather than fungicidal, sprays or dusts.

CONTROL OF BROWN ROT DISEASES: GENERAL PRINCIPLES

Methods of control applicable to special conditions have already been discussed in relation to the various diseases described, for the measures to be adopted must depend on the host plant, the mode of infection and the organs attacked. There are, however, certain general principles involved and these are worthy of consideration here.

The value of sanitary measures as a means of removing sources of infection has been repeatedly stressed, for the primary infections in any one year are brought about by spores shed from the fungus fructifications developing on fruit, flowers, leaves, twigs, spurs and branches which had become infected during the previous year. It has been mentioned also that infection results in the rapid death of the organs attacked, and in the appearance at the surface, within a very short time, of numerous Monilia fructifications. The conidia are readily dispersed and, falling on a suitable substratum, repeat the process so that there are several generations of Monilia fructifications during the course of the summer. The earlier in the season any infected parts are detected and removed, therefore, the more likely are these sanitary measures to prove effective.

The great variability in the degree of incidence of these diseases from year to year should be borne in mind, for it is important that they should not be ignored even in years when they appear to be almost absent. Severe outbreaks have frequently occurred following years when there was little noticeable infection. In such cases the result was brought about by weather conditions particularly favourable for the fungi, together with the presence of relatively few sources of infection which were left on the trees from the previous year. With apple blossom wilt, it has been shown that a few dead spurs left on the trees may be quite sufficient to account for the death of very many new trusses of flowers. Plum trees on which almost all the blossom has been destroyed one year have been known to have been only very slightly infected in the previous year, so little indeed that the grower had not taken the trouble to deal with the matter. On Morello cherries too, a few dead twigs left on a tree may cause dozens of twigs to be killed the following year and two or three years of neglect may render the trees almost or completely barren in a bad " Brown Rot year ".

The cutting out of infected spurs, twigs and branches should be carried out, if at all possible, immediately after the disease is noticed. At that time there is little risk of overlooking any infections as the wilted flowers and leaves indicate clearly where they are. If the operation is delayed until later in the summer, the trees by that time may have made so much growth that many infected twigs will be hidden. Treatment is sometimes postponed until the winter when the normal pruning is carried out, but small dead twigs and spurs are not then easily distinguished and a sufficient number of them may be left to be a source of trouble in the following season. Mummified fruits, however, are conspicuous during the winter and these should all be removed and destroyed before the trees resume growth.

Ornamental shrubs and bushes of wild susceptible rosaceous species may serve as sources of infection for fruit trees and if such shrubs are in the vicinity, they should be examined and any infected parts removed and burnt (see p. 57).

In Europe, mummified fruits which fall to the ground usually disintegrate during the winter and, as already stated, the perfect stages of the brown rot fungi have been found on only very few occasions. Where *S. fructicola* abounds, however, the apothecial fructifications developing from the mummies are a real danger and any mummies lying on the ground should be ploughed-in deeply.

Since primary infection of the fruit usually occurs through definite injuries, particularly those caused by insects, the control of insect pests indirectly aids in the control of brown rot. The value of suitable routine spraying against insects and scab has been mentioned in relation to apple brown rot.

In most cases, the sanitary measures outlined above must be supplemented by applications of fungicides (see p. 49). The value of winter washing, designed to destroy the Monilia fructifications as they develop in spring on the organs in which the fungus has overwintered, is doubtful, though some measure of success has been obtained in certain cases with tar-distillate, caustic washes and other eradicant sprays. Spraying before and after flowering must be regulated by a consideration as to whether the blossom or fruit or both are to be protected. When blossom wilt is a primary trouble, one or two pre-blossom sprays or sprays in bloom should be applied. If infection through unopen flowers is expected (as in Morello cherry and apricot), the first application should be made while the petals are still enveloped by the calyx. For infection through the open flowers, as in most cases of blossom wilt, a pre-blossom application should be made just when the petals are showing between the calyx lobes (e.g. pink bud stage in peaches). It has been claimed that good results have been obtained by spraying when the petals have expanded, particularly on peach trees in the United States, but as there is some risk of scorching the petals, the method should be adopted with caution unless the trees have flowers in abundance; the destruction of some of the flowers by the spray may be compensated for by greater freedom from infection of the flowers which open later.

Spraying or dusting after flowering is carried out with the object of protecting the fruit and several applications may be necessary, especially if the fruit is to be transported long distances.

In packing and storing, care must be taken to select only sound fruit, for not only is there the possibility that the damaged fruits will develop brown rot but they may transmit it to others by contact. Large soft fruits, such as peaches, which are to be sent long distances, should be wrapped in paper. It may be found necessary to disinfect the packing cases if these have been used previously for fruit which has rotted. This may be done by immersing them for one minute in boiling water or in formalin solution 1:320, and then allowing them to dry before they are used again for packing fruit.

It may be well to emphasize again that to neglect brown rot in a year when the damage is slight is a most unsound policy, for this neglect will almost certainly result in far greater loss in the following year, and in addition, in the expenditure of a much greater amount of labour in the removal of infected parts when this becomes imperative, as it eventually will.

Breeding to raise fruit trees resistant to brown rot has not yet received much attention, probably because such work with fruit trees (unlike annual plants) involves very long-term projects and new hybrid varieties may require many years before they are old enough to show evidence of their resistance or susceptibility. It is common knowledge, however, that in fruit species there are certain varieties more resistant than others and genetical studies of such varieties might afford a guide to breeding experiments. The problem has been considered by some investigators, as for instance Zaprometoff (1925) who finds that there are species of apple and pear resistant to or immune from brown rot, which might be of use in breeding experiments. Michurin claimed that some of his hybrid varieties of apple did not become attacked by fruit brown rot but these observations require confirmation under controlled conditions. Some success in the United States in raising resistant varieties of apricots is mentioned by Kotte (1939), and further work in this direction may be expected in the future.

BIBLIOGRAPHY

References mentioned in the text are marked with an asterisk.

Titles of papers given in brackets are translations and where appropriate the reference to an abstract is also given, (for instance an abstract in *Rev. appl. Mycol.* appears thus [*R.A.M.*—,—]).

*Adam, D. B. (1923). Experiments in the storage of fruits. *J. Dep. Agric. Vict.*, **21**, 178–186; 234–241; 371–382.

*Adam, D. B. (1924). Soft fruits storage experiments. *Fruit World, Melbourne*, **25**, 73–74.

*Adam, D. B. (1935). Summary of plant disease records in South Australia for two years ending 30th June, 1934. *J. Dep. Agric. S. Aust.*, **38**, 939–942.

Adam, J. F. (1924). Plant diseases and their prevalence for 1923 in Delaware. *Exten. Circ. Del. agric. Exp. Sta.*, 14, 29 pp.

Aderhold, R. (1897). Zur Monilia—Epidemie der Kirschbaume. *Gartenflora*, pp. 429–433.

Aderhold, [R.] (?1898). Ueber die in den letzten Jahren in Schlesien besonders hervorgetretenen Schäden und Krankheiten unseren Obstbäume und ihre Beziehungen zum Wetter. *Sitzung Sect. Obst- u. Gartenbau*, Dec., 1897, 27 pp.

Aderhold, R. (1902). Die Monilia-Krankheiten unserer Obstbäume und ihre Bekämpfung. *Flugbl. biol. Reichsanst., Berl.*, 14, 4 pp.

Aderhold, R. (1904). Über eine vermuthliche zu *Monilia fructigena* Pers. gehörige Sclerotinia. (Vorläufige Mitteilung). *Ber. dtsch. bot. Ges.*, **22**, 262–266.

*Aderhold, R., & Ruhland, W. (1905). Zur Kenntnis der Obstbaum-Sklerotinien. *Arb. biol. Abt. (Anst.-Reichsanst.), Berl.*, **4**, 427–442.

*Ainsworth, G. C., & Bisby, G. R. (1950). *A dictionary of fungi.* 3rd Edition, pp. 411–427. London.

*Albertini, J. B. de, & Schweinitz, L. D. de. (1805). *Conspectus Fungorum in Lusatiae superioris Agro Niskiensi crescentium*, No. 1090, p. 365, Lipsiae.

*Alcock, N. L., & Foister, C. E. (1931). List of fungous diseases received by the Pathological Department of the Department of Agriculture for Scotland. *Trans. Proc. bot. Soc. Edin.*, **30**, 338–350.

*American Phytopath. Soc. Comm. on Standardization of Fungicidal Tests. (1943*a*). The slide-germination method of evaluating protectant fungicides. *Phytopathology*, **33**, 627–632.

American Phytopath. Soc. Comm. on Standardization of Fungicidal Tests. (1943*b*). Standard Laboratory Bordeaux Mixture. *Phytopathology*, **33**, 633–634.

*Ames, Adeline. (1915). The temperature relations of some fungi causing storage rots. *Phytopathology*, **5**, 11–19.

*Anagnostopoulos, P. Th. (1930). The cause of unfruitfulness and withering of almonds in Greece. *Minist. Agric. Athens*, 14 pp.

*Anagnostopoulos, P. Th. (1933). The gummosis of the drupaceous fruit trees. The causes and the means of control. *Sup. Seh. Agric. Athens, Greece*, 40 pp.

*Anderson, H. W. (1925*a*). Observations on fruit diseases in 1924. *Trans. Ill. hort. Soc.*, **58**, 243–255.

(Anderson, H. W.). (1930). A year's progress in solving farm problems of Illinois. *Ann. Rep. Ill. agric. Exp. Sta. for year ended June* 30, 1930. 296 pp.

*Anderson, H. W. (1933). Results of disease control in 1932. *Trans. Ill. hort. Soc.*, **66**, 175–200.

*Andreyeff, N. I. (1924). [Fungus parasites in the Don Province]. *Pamph. N. Caucasus reg. agric. Admin. Rostoff-on-Don.* 27 pp. [*R.A.M.*, **4**, 398].

*Angell, H. R. (1949). Observations on brown rot of stone fruits in four successive seasons in four Canberra suburbs. *J. Aust. Inst. agric. Sci.*, **15**, 159–160.

Angell, H. R. (1950). Some instances of great variability in incidence of brown rot of stone fruits in Canberra in 1949–50. *J. Aust. Inst. agric. Sci.*, **16**, 33–34.

Anon. (1924). Bericht der höheren staatlichen Lehrenstalt für Obst- und Gartenbau zu Proskan für die Rechnungsjahr 1922 und 1923. *Landw. Jahrb.*, **60**, 519–543.

Anon. (1926*a*). Spray calendar for plums and cherries. *Circ. N. J. agric. Exp. Sta.*, 182, 4 pp.

Anon. (1926*b*). Control of brown-rot in stone fruits. Recent experiments at Henderson. *N.Z. J. Agric.*, **33**, 170–173.

*Anon. (1933). Brown rot of stone fruits. *Fruit World, Melbourne*, **34**, 335–336.

*Anon. (1935). Protecting plants from disease. *Rep. Wis. agric. Exp. Sta.* 1933–1934, pp. 15–32.

*Anon. (1945). Brown rot (*Sclerotinia fructicola*). Cherry, peach, apricot, nectarine, and plum. *J. Dep. Agric. S. Aust.*, **49**, 210–211.

*Anrès, M. & Joëssel, P. H. (1932). Observations sur l'apparition et le dévelopement du Monilia de l'Abricotier en 1931 et 1932 dans les communes de Saint-Hyppolite, Caromb et Le Barroux (Vaucluse). Méthode de lutte employée et résultats obtenus. *Rev. Path. veg.*, **19**, 248–252.

*Arnstein, H. R. V., Cook, A. H. & Lacey, M. S. (1948). The inhibition of *Fusarium oxysporum* var. *cubense* by musarin, an antibiotic produced by Meredith's actinomycete. *J. gen. Microbiol.*, **2**, 111–122.

*Arthur, J. C. (1886). Rotting of cherries and plums. *Oidium fructigenum* S. & K. *Rep. N.Y. agric. Exp. Sta. for* 1885, **4**, 254–258.

Babel, A. (1935). Infrarot-Photographie im Pflanzenschutz. *Angew. Bot.*, **17**, 43–53.

*Bailey, J. S. (1948). Further observations of mummy berry on cultivated blueberries. *Proc. Amer. Soc. hort. Sci.*, **52**, 299–303.

*Balakhonoff, P. I. (1930). [Blossom Wilt of fruit trees on the Black Sea coasts in relation to fogs.] *Bull. N. Caucasian Plant Prot. Sta.*, 6–7, 169–172. [R.A.M., **10**, 605.]

Balakhonoff, P. I. (1931*a*). The question of crop grading. The cause of storage rots of apples. *Pl. Prot., Leningr.*, **8**, pp. 35–37.

*Balakhonoff, P. I. (1931*b*). The severe development of Monilia Blossom Wilt of stone fruits in Northern Caucasia. *Mater. Mikol. Fitopat. Ross.*, **8**, 137–139.

*Baldwin, M. M. (1950). Sulfur in fungicides. *Industr. Engng Chem.*, **42**, 2227–2230.

*Barker, B. T. P. (1928). Investigations on the fungicidal action of sulphur. Progress report. *Rep. agric. hort. Res. Sta. Bristol*, 1927, 72–80.

*Barker, B. T. P., Gimingham, C. T. & Wiltshire, S. P. (1920). Sulphur as a fungicide. *Rep. agric. hort. Res. Sta. Bristol*, 1919, 57–75.

Barnett, H. L. (1944). Stone fruit diseases in central California. *Plant Dis. Reptr*, **28**, 424–425.

Barnett, H. L. & Bodine, E. W. (1944). Results of the brown rot survey, and notes on some other diseases of stone fruits in central California. *Plant Dis. Reptr*, **28**, 181–183.

*Barnett, H. L. & Lilly, V. G. (1947). Vitamin deficiencies in the Sclerotiniaceae. (Abstr.) *Phytopathology*, **37**, 2.

*Barss, H. P. (1923). Brown-rot and related diseases of stone fruits in Oregon. *Circ. Ore. agric. Exp. Sta.*, **53**, 18 pp.

Barss, H. P. (1925). Serious blossom blight in Pacific Northwest orchards due to a species of Monilia. (Abstr.) *Phytopathology*, **15**, 126.

Barss, H. P. (1930). European and American brown-rot blossom blight in western Oregon. (Abstr.) *Phytopathology*, **20**, 855.

*Baur, K. & Huber, G. A. (1940). The use of calcium cyanamid and other fertilizer materials and soil amendments in the destruction of apothecia of *Sclerotinia fructicola* with methods of application. (Abstr.) *Phytopathology*, **30**, 785

Baur, K. & Huber, G. A. (1941). Effect of fertilizer materials and soil amendments on development of apothecia of *Sclerotinia fructicola*. *Phytopathology*, **31**, 1023–1030.

*Bazán de Segura, Consuelo. (1946). La podredumbre morena del Melocotonero en el Perú. *Bol. Estac. exp. agríc. La Molina*, 30, 13 pp. [R.A.M., **27**, 327.]

*Behrens, J. (1898). Beiträge zur Kenntnis der Obstfaulnis. *Zbl. Bakt.*, **4**, 514 et seq.

Behrens, J. (1899). Kupferpräparate und *Monilia fructigena*. *Zbl. Bakt.*, **5**, 507–509.

*Benlloch, M. (1931). El moho de las frutas. *Bol. Pat. veg. Ent. agric. Madr.*, **5**, 134–136.

*Bennett, S. H., Kearns, H. G. H. & Marsh, R. W. (1945). A field trial on the combined control of codling moth and brown rot of apples. *Rep. agric. hort. Res. Sta. Bristol*, 1944, 157–161.

*Berkeley, G. H. (1927). Report of the Dominion Field Laboratory of Plant Pathology, St. Catherines, Ontario. *Rep. Dom. Botanist for* 1926, *Div. Bot. Canad. Dep. Agric.*, pp. 59–102.

*Berkeley, G. H. (1928). Blossom blight on peaches. *Rep. Dom. Botanist for* 1927, *Div. Bot. Canad. Dep. Agric.*, p. 123.

*Berkeley, G. H. (1929). The brown rot of stone fruits. *Sixtieth ann. Rep. Fruit Grs' Ass. Ontario*, 1928, pp. 50–54.

Berkeley, G. H. & Willison, R. S. (1944). Brown Rot on stone fruits—Prevention and Control. *Dep. Agric. Ottawa Canad. Div. Bot. Pl. Path. Contr.*, 769, 3 pp.

*Berkeley, M. J. (1836). *The English Flora of Sir James Edward Smith.* Vol. **5**, part II, p. 349. London.

*Berkeley, M. J. (1857). *Introduction to Cryptogamic Botany*, p. 308. London.

Berkeley, M. J. (1860). *Outlines of British Fungology*. London.

*B(erkeley), M. J. (1864). [Brown rot of apricots]. *Gdnrs' Chron.*, **35**, 818.

*Berkhout, Christine M. (1923). De Schimmelgeslachten Monilia, Oidium, Oospora en Torula. Thesis, Scheveningen.

*Bevilacqua, I. (1935). Una grave infezione nei Ciliegi. *Istria agric.*, **15**, 252–254.

*Biological Branch, N.S.W. Department of Agriculture, (1941*a*). New Plant diseases. *Agric. Gaz. N.S.W.*, **52**, 163.

Biological Branch, N.S.W. Department of Agriculture. (1941*b*). Brown rot of early apricots. *Agric. Gaz. N.S.W.*, **52**, 579–581.

Biological Branch, N.S.W. Department of Agriculture. (1941*c*). Cherry Brown rot. *Agric. Gaz. N.S.W.*, **52**, 644–645.

Blodgett, E. C. (1950). Tree fruit diseases in western Washington. *Plant Dis. Reptr*, **34**, 147–148.

*Blumer, S. & Kundert, J. (1950). Methoden der biologischen Laborprüfung von Kupferpräparaten. *Phytopath. Z.*, **17**, 161–199.

Blunck, H. (1944). Verluste an Haselnüssen durch Monilia-Befall. *Z. PflKrankh.*, **54**, 332–334.

*Bondartzev, A. S. (1931). [*Diseases of cultivated plants and their control.*] pp. 366–372. Leningrad.

*Bonorden, H. F. (1851). *Handbuch der allgemeinen Mykologie*, p. 76. Stuttgart.

Bonsmann, M. R. & Thomas, B. (1947). Die Verwertung von schwarzfaulen Äpfeln. *Pharmazie, Berlin*, **2**, 557–559.

*Boudier, E. (1907). *Histoire et Classification des Discomycetes d'Europe*, pp. 108–109. Paris.

*Bouhelier, R. (1936). Traitements en hiver. *Fruits Primeurs*, **6**, 341–345.

*Boyle, C., Murphy, M. & Cummins, H. A. (1928). " Blossom-wilt " of apple trees and " Wither-tip " of plum trees, with special reference to two biologic forms of *Monilia cinera* Bon. *Sci. Proc. R. Dublin Soc.*, **19**, 63–76.

Bratley, C. O. (1931). Decay of sweet cherries from California. *Plant Dis. Reptr*, **15**, 73–74.

*Bratley, C. O. & Wiant, J. S. (1950). Diseases of fruits and vegetables found on the market, and means of controlling them. *Econ. Bot.*, **4**, 177–191.

*Braun, K. (1928). Die Obstscheunen des " Alten Landes " und ihre Schädlinge. *Mitt. Ges. Vorratsschutz*, **4**, 5–6, 18–19.

Braun, K. (1933). Obstfäulnis bei Aepfeln und ihre Verhütung. *Landw. Wochenbl. Schleswig-Holstein*, **83**, 31, 33, 35, 37. [*R.A.M.*, **13**, 35].

*Bremer, H., Işmen, H., Karel, G., Özkan, H. & Özkan, M. (1947). Beiträge zur Kenntnis der parasitischen Pilze der Türkei. I. *Rev. Fac. Sci. Univ. Istanbul*, **13**, 122–172. [*R.A.M.*, **26**, 533].

*Brierley, P. (1950). Eradicant fungicides of possible value against the azalea petal blight fungus, *Ovulinia azaleae*. *Phytopathology*, **40**, 153–155.

*Britton, W. E., Zappe, M. P. & Stoddard, E. M. (1922). Experiments in dusting versus spraying on apples and peaches in Connecticut in 1921. *Bull. Conn. agric. Exp. Sta.*, 235, 17 pp.

*Brooks, C. (1906). Temperature and toxic action. *Bot. Gaz.*, **42**, 359–375.

*Brooks, C. (1933). Spoilage of stone fruits on the market. *Circ. U.S. Dep. Agric.*, 253, 11 pp.

*Brooks, C., Bratley, C. O. & McColloch, L. P. (1936). Transit and storage diseases of fruits and vegetables as affected by initial carbon dioxide treatment. *Tech. Bull. U.S. Dep. Agric.*, 519, 24 pp.

*Brooks, C. & Cooley, J. S. (1917). Temperature relations of apple-rot fungi. *J. agric. Res.*, **8**, 139–164.

Brooks, C. & Cooley, J. S. (1921). Temperature relations of stone fruit fungi. *J. agric. Res.*, **22**, 451–465.

*Brooks, C. & Cooley, J. S. (1928). Time-temperature relations in different types of peach-rot infection. *J. agric. Res.*, **37**, 507–543.

Brooks, C., Cooley, J. S. & Fisher, D. F. (1930). Diseases of apples in storage. *Fmrs' Bull. U.S. Dep. Agric.*, 1160, 23 pp.

Brooks, C. & Fisher, D. F. (1916). Brown-rot of prunes and cherries in the Pacific Northwest. *Bull. U.S. Dep. Agric.*, 368, 10 pp.

*Brooks, C. & Fisher, D. F. (1921). Transportation rots of stone fruits as influenced by orchard spraying. *J. agric. Res.*, **22**, 467–477.

*Brooks, C. & Fisher, D.F. (1924). Prune and cherry brown-rot investigations in the Pacific Northwest. *Bull. U.S. Dep. Agric.*, 1252, 21 pp.

Brooks, C. & Fisher, D. F. (1925). Spraying for brown rot in the Northwest. *Amer. Fruit Gr.*, **45**, 10, 25, 34.

*Brooks, C., Miller, E. V., Bratley, C. O., Cooley, J. S., Mook, P. V. & Johnson, H. B. (1932). Effect of solid and gaseous carbon dioxide upon transit diseases of certain fruits and vegetables. *Tech. Bull. U.S. Dep. Agric.*, 318, 59 pp.

*Brown, W. (1922). On the germination and growth of fungi at various temperatures and in various concentrations of oxygen and of carbon dioxide. *Ann. Bot., Lond.*, **36**, 257–283.

*Brož, O. (1913). Die Moniliagefahr. *Der Obstzuchter*, Nr. 7. 3 pp.

*Buchwald, N. F. (1936). Plantepatologiske Meddeleleser 1–5. *K. Vet-Højsk. Aarsskr. 1936*, 132–140.

*Buchwald, N. F. (1943). Paavisning av *Monilinia* (*Sclerotinia*) *fructigena* (Aderh. et Ruhl.) Honey paa Hasselnød (*Corylus avellana*). *Tidsskr. Planteavl*, **47**, 521–538.

*Buchwald, N. F. (1949). [Studies in the Sclerotiniaceae. I. Taxonomy of the Sclerotiniaceae.] *K. Vet. Højsk. Aarsskr. 1949*, pp. 76–191.

Bucksteeg, W. (1939*a*). Über die Monilia-Anfälligkeit unserer Obstsorten. *Z. PflKrankh.*, **49**, 11–15.

*Bucksteeg, W. (1939*b*). Untersuchungen über den Sporenflug bei Monilia als Grundlage für die chemische Bekämpfung. Vorläufige Mitteilung. *Z. PflKrankh.*, **49**, 252–258.

*Bucksteeg, W. (1940). Untersuchung über die Wirkung von Kältegraden auf Keim- und Infektionsfähigkeit der Konidien von *Sclerotinia cinerea* Schroet. und *Sclerotinia fructigena* Schroet. *Z. PflKrankh.*, **50**, 507–512.

Bullock, F. J. (1941). Brown rot in peaches. Control methods described. *Fruit World, Melbourne*, **42**, 10.

*Bureau de la Défense des Végétaux, Maroc. (1941). Les Maladies du Pécher et de l'Amandier. *Mémento Dir. Prod. agric. Maroc*, 60, 17 pp.

*Burkholder, C. L. (1943). Plum spraying problems. *Hoosier Hort.*, **25**, 52–54.

*Butler, E. J. & Bisby, G. R. (1931). The fungi of India. *Imper. Counc. agric. Res., Scient. Monograph*, 1, p. 13.

*Byrde, R. J. W. (1949). Experiments on the control of brown rot of fruits: progress report 1947–48. *Rep. agric. hort. Res. Sta. Bristol, 1948*, 161–173.

*Byrde, R. J. W. (1950). Experiments on the control of brown rot of fruits: progress report 1948–49. *Rep. agric. hort. Res. Sta. Bristol, 1949*, 81–89.

*Byrde, R. J. W. (1952). Experiments on the control of brown rot of apples and plums. I. Laboratory tests. *J. hort. Sci.*, **27**, 130–144.

Byrde, R. J. W. (1952). The effect of age of wound and of weather on the susceptibility of apple injuries to infection by the brown rot fungus. *Rep. agric. hort. Res. Sta. Bristol, 1951*, pp. 128–131.

Byrde, R. J. W. & Fielding, A. H. (1952). Tests of eradicant fungicides against brown rot. I. *Rep. agric. hort. Res. Sta. Bristol, 1951*, pp. 131–133.

*Calavan, E C. (1944). The use of eradicant and blossom sprays on sour cherry in the control of brown-rot and spur blight incited by *Sclerotinia laxa* Ader. and Ruhl. (Abstr.) *Phytopathology*, **34**, 997.

Calavan, E. C. & Keitt, G. W. (1948). Blossom and spur blight (*Sclerotinia laxa*) of sour cherry. *Phytopathology*, **38**, 857–882.

*Carpenter, J. B. (1942). A toximetric study of some eradicant fungicides. *Phytopathology*, **32**, 845–856.

*Cardinell, H. A. & Mitchell, A. E. (1948). Packing house trials to reduce peach-rot *Quart. Bull. Mich. agric. Exp. Sta.*, **30**, 460–467.

Carter, J. C. (1935). Growth association of microorganisms. (Abstr.) *Phytopathology*, **25**, 9.

Cation, D. & Dunegan, J. C. (1949). The overwintering of *Monilinia fructicola* in twig cankers under Michigan conditions. *Plant Dis. Reptr*, **33**, 97–98.

Cation, D., Dunegan, J. C. & Kephart, J. (1949). The occurernce of *Monilinia laxa* in Michigan. *Plant Dis. Reptr*, **33**, 96.

*Cayley, D. M. (1915). Brown rot of fruit. *Gdnrs' Chron.*, **58**, 269–270.

*Chaboussou, —. (1945). Sur deux rynchites (*R. bacchus* L. et *R. aequatus* L.) nuisibles aux arbres fruitiers en Agenais. *C.R. Acad. Agric. Fr.*, **31**, 110–112.

*Chabrolin, C. (1922). Le dépérissement des Abricotiers dans la Vallée du Rhône. *Vie agric. rur.*, **20**, 415–416.

*Chabrolin, C. (1923*a*). Les maladies des arbres fruitiers à noyaux dans la Vallée du Rhône. *Off. rég. agric. Midi. Bull. trimest.* No. 6, pp. 1–27.

*Chabrolin, C. (1923*b*). Observations sur les Maladies de l'abricotier. II. Monilia de l'abricotier (*Sclerotinia cinera*). *Off. rég. agric. Midi. Bull. trimest.* No. 6, pp. 13–18.

*Chabrolin, C. (1924). Quelques maladies des arbres fruitiers de la Vallée du Rhône. *Ann. Épiphyt.*, **10**, 286–308.

Chester, K. S. (1942). *The nature and prevention of plant diseases*, pp. 138–143. Philadelphia.

Chifflot, J. (1917). Principales maladies cryptogamic des arbres fruitiers a noyaux dans le Rhône et la vallée du Rhône. *Pomol. franç.* No. 1, pp. 27–32.

*Chifflot, J. (1921). Les maladies cryptogamiques des Abricotiers dans la vallée du Rhône. *Ann. Épiphyt.*, **7**, 315–322.

*Christoff, A. (1934*a*). [Some plant diseases new to Bulgaria (II. Contribution)]. *Bull. Soc. Bot. de Bulgarie*, **6**, p. 13.

*Christoff, A. (1934*b*). [Apparatus for catching wind-borne spores.] *J. agric. Exp. Stas. Bulgaria*, **6**, 41–48.

*Christoff, A. (1938). The brown rot diseases of fruit trees in Bulgaria. *J. agric. Exp. Stas. Bulgaria*, **8**, 3–32.

*Chrzaszy, T. & Schillak, P. (1936). Die Umbildung der Milchsäure durch verschiedene. Schimmelpilze. *Biochem. Zbl.*, **288**, 359–368. [*Zbl. Bakt.*, **96**, 169].

Ciferri, R. (1927). Notas mycologicae et phytopathologicae. Serie II. N. 1–15. *Riv. Patol. veg.*, **17**, 209–294.

*Ciferri, R. (1928). Micoflora Domingensis. Lista de los hongos hasta la fecha indicadoes en Santa Domingo. *Estac. agron. Moca, Rep. Dominica*, Ser. B.—Botanico No. 14 Dic. 1928, 260 pp.

*Ciferri, R. (1950). [The fungicidal power of hexanitrodiphenylamine and its synergism with copper salts.] *Not. Mal. Piante*, No. 9, pp. 40–43. [*R.A.M.*, **29,** 471].

*Ciferri, R. & Parodi, E. (1933). Descrizione del fungo che causa la " Moniliasi " del cacao. *Phytopath. Z.*, **6,** 539–542.

*Ciferri, R. & Scaramuzzi, G. (1947). Prove dirette di un' efficacia fungistatica di complessi zinco- e cupro-ossido-chinoliniei. *Atti Ist. bot. Univ. Pavia*, **3,** 257–260. [*R.A.M.*, **27,** 484].

*Ciferri, R. & Scaramuzzi, G. (1949). Azione fungistatica della tirotriticina sulla germinazione di conidi fungini e possibilita d'un suo dosaggio biologico per tale via. *Atti Ist. Bot. Univ. Pavia*, **3,** 282–285.

*Clayton, C. N. (1942). The germination of fungous spores in relation to controlled humidity. *Phytopathology*, **32,** 921–943.

Clinton, G. P. (1934). Plant pest handbook for Connecticut. II. Diseases and injuries. *Bull. Conn. agric. Exp. Sta.*, 358, 154–329.

*Cohen, S. I. (1951). Effectiveness of three phenyl mercury formulations for the control of apple scab. (Abstr.) *Phytopathology*, **41,** 657.

*Cohen, V. L. (1942). The content of fungous spores in the air in Buffalo, New York. (Abstr.) *J. Bact.*, **43,** 115–116. [*R.A.M.*, **21,** 290].

*Comes, O. (1891). *Crittogamia agraria*, p. 207. Naples.

Conners, I. L. & Savile, D. B. O. (1944). *Twenty-third Annual Report of the Canadian Plant Disease Survey*, 1943, 122 pp. Department of Agriculture, Canada.

Cook, M. T. (1913). Report of the Plant Pathologist for the year 1912. *N.J. agric. Coll. Exp. Sta.*, pp. 509–527.

Cook, M. T. (1921*a*). Brown rot of the peach. *N.J. agric. Coll. Exp. Sta. for the year ending June* 30, 1920, p. 573.

Cook, M. T. (1921*b*). The blossom blight of the peach. *Phytopathology*, **11,** 290–294.

Cook, M. T. (1923). Prown rot of apple. *Phytopathology*, **13,** 462.

*Cooke, M. C. (1871). *Handbook of British Fungi*, pp. 603–604. London.

*Cooke, M. C. (1906). *Fungoid Pests of Cultivated Plants*, pp. 135–137. London.

*Cooley, J. S. (1914). A study of the physiological relations of *Sclerotinia cinerea* (Bon.) Schröter. *Ann. Mo. bot. Gdn.*, **1,** 291–326.

*Cristinzio, M. (1934). Malattie delle piante da frutta nella Campania e nel Mezzogiorno. *R. Lab. Pat. Veg. Portici*, **3,** 47–87.

*Crocker, W. (1948). *Growth of Plants. Twenty Years' Research at Boyce Thompson Institute*, pp. 459. New York.

Crosby, C. R., Mills, W. D. & Blauvelt, W. E. (1931). Protecting orchard crops from diseases and insects. *Bull. Cornell agric. Exp. Sta.*, 498, 78 pp.

*Crowell, I. H. & Lavalee, E. (1942). *Check list of diseases of economic plants in Canada*. 66 pp. *Dominion Department of Agriculture, Canada.*

*Croxall, H. E., Collingwood, C. A. & Jenkins, J. E. E. (1951). Observations on brown rot (*Sclerotinia fructigena*) of apples in relation to injury caused by earwigs (*Forficula auricularia*). *Ann. appl. Biol.*, **38,** 833–843.

*Culpepper, C. W. & Moon, H. H. (1920). Sulphur-spray residues and the swelling of tin cans packed with peaches. *J. agric. Res.*, **39,** 31–40.

*Cunningham, G. H. (1922*a*). Brown rot, *Sclerotinia cinerea* Schroet. Its appearance, cause and control. *N.Z. J. Agric.*, **24,** 83–98.

*Cunningham, G. H. (1922*b*). Occurrence of apothecia of brown rot in New Zealand. *N.Z. J. Agric.*, **25,** 177.

*Cunningham, G. H. (1922*c*). The significance of apothecia in the control of brown-rot of stone-fruits. *N.Z. J. Agric.*, **25,** 225–230.

*Cunningham, G. H. (1922*d*). The second-hand fruit case as a source of fungous disease infection. *N.Z. J. Agric.*, **24,** 241–243.

*Cunningham, G. H. (1925*a*). Brown-rot, *Sclerotinia cinerea* Schroet. Its appearance and control. *Bull. N.Z. Dep. Agric.*, 101, 11 pp.

*Cunningham, G. H. (1925*b*). *Fungous diseases of fruit-trees and their remedial treatment*, pp. 228–242. Auckland, New Zealand.

*Cunningham, G. H. (1934). Orchard sprays in New Zealand. *N.Z. J. Agric.*, **48,** 1–12.

Cunningham, G. H. (1949). Therapeutant testing. Brown-rot of peaches. *Ann. Rep. Dep. sci. industr. Res. N.Z.*, p. 52.

*Curtis, K. M. (1928). The morphological aspect of resistance to brown rot in stone fruit. *Ann. Bot., Lond.*, **42,** 39–68.

*Curtis, K. M. (1929). Peach culture. I. Four years' control of brown rot in New Zealand. *Res. Bull. Dep. sci. industr. Res., N.Z.* 15, pp. 4–17.

*D'Aeth, H. R. X. (1937). The interaction of two apple-rotting fungi. (Abstr.) *Phytopathology*, **27,** 126.

*Daines, R. H. (1942). Brown rot of peach and its control. *Circ. N. J. agric. Exp. Sta.* 434, 8 pp.

Dallas, W. K. (1937). Spray schedule for control of principal orchard diseases and pests. *N.Z. J. Agric.*, **54**, 15–24.

*Darnell-Smith, G. P. & Mackinnon, E. (1915). Fungus and other diseases of stone fruits. *Agric. Gaz. N.S.W.*, **26**, 591–598.

*Darpoux, H. & Faivre-Amiot, A. (1949*a*). Actions antagonistes de l'Actinomycète 105 sur quelques micro-organismes pour la plupart phytopathogènes. Essais d'application dans la lutte contre les maladies des plantes. *Ann. Inst. Pasteur*, **76**, 72–74. [*R.A.M.*, **30**, 58].

*Darpoux, H. & Faivre-Amiot, A. (1949*b*). Actions antagonistes de divers micro-organismes sur les agents phytopathogènes. *C. R. Acad. Agric. Fr.*, **35**, 266–269.

*Davison, F. R. & Willaman, J. J. (1927). Biochemistry of plant diseases. IX. Pectic enzymes. *Bot. Gaz.*, **83**, 329–361.

*Delacroix, G. & Maublanc, A. (1909). *Maladies des plantes cultivées. 2. Maladies parasitaires*, pp. 260–263. Paris.

Delhaye, R. ( ). Moniliose ou rot brun du pommier et du poirier, *Monilia fructigena* Pers. *Courr. hort.*, **7**, 102.

Demaree, J. B. & Wilcox, M. S. (1947). Fungi pathogenic to blue-berries in the eastern United States. *Phytopathology*, **37**, 487–506.

*Dementyeva, M. I. (1951). [Increasing disease resistance in the apricot by pruning.] *Sad i Ogorod*, 1951, No. 3, pp. 32–34.

*Denham, H. (1946). Apricots. Some further observations. *J. R. hort. Soc.*, **71**, 101–104.

*Denham, H. & Wormald, H. (1942). The brown rot diseases of the apricot. *J. R. hort. Soc.*, **67**, 261–263.

*Dennis, R. W. G. & Foister, C. E. (1942). List of diseases of economic plants recorded in Scotland. *Trans. Brit. mycol. Soc.*, **25**, 266–306.

Department of Botany and Plant Pathology. (1930). *Bienn. Rep. Oregon agric. Exp. Sta.* 1926–28, pp. 78–82.

*Dickson, H. (1939). The effect of the growth of *Sclerotinia fructigena* of alternating periods of light and darkness of equal length. *Ann. Bot., Lond.*, **3**, 131–136.

*Dillon Weston, W. A. R. (1945). Brown rot of apples. *J. Minist. Agric.* **52**, 135–137.

*Dillon Weston, W. A. R. & Petherbridge, F. R. (1933). Apple and pear scab in East Anglia. *J. Pomol.*, **11**, 185–198.

Dimond, A. E. & Horsfall, J. G. (1944). Synergism as a tool in the conservation of fungicides. *Phytopathology*, **34**, 136–139.

Dodge, B. O. & Rickett, H. W. (1943). *Diseases and pests of ornamental plants*, pp. 638. Lancaster, Pennsylvania.

*Doidge, Ethel M. (1919). Brown rot of fruit mould. *S.A. Fruit Gr.*, Nov. 1919.

*Doidge, Ethel M. (1950). The South African fungi and lichens to the end of 1945. *Bothalia*, **5**, 222; 837; 838.

*Doidge, Ethel M. & Bottomley, A. M. (1931). A revised list of plant diseases occurring in South Africa. *Botanical Survey of S. Africa*, *Mem.* 11, 78 pp.

*Doran, W. L. (1922). Effect of external and internal factors on the germination of fungous spores. *Bull. Torrey bot. Cl.*, **49**, 313–336.

*Dowson, W. J. (1925). A core rot of apples. *Gdnrs' Chron.*, **78**, 479–480.

*Dowson, W. J. (1926). On a core rot and premature fall of apples associated with *Sclerotinia fructigena*. *Trans. Brit. mycol. Soc.*, **11**, 155–161.

*Dowson, W. J. & Dillon Weston, W. A. R. (1937). Brown rot of the hawthorn. *Gdnrs' Chron.*, **101**, 426.

*Drayton, F. L. (1932). The sexual function of the microconidia in certain discomycetes. *Mycologia*, **24**, 345–348.

*Drummond, R. (1934). Notes on the wither-tip disease of plums caused by *Sclerotinia cinerea* and on the blackening of apples caused by *Sclerotinia fructigena*. *J. Pomol.*, **12**, 105–109.

*Dunegan, J. C. (1940). A blight of wild cherry seedlings. *Phytopathology*, **30**, 89–90.

*Dunegan, J. C. (1947). The occurrence of *Monilinia seaveri* on English Morello cherry. *Phytopathology*, **37**, 929–930.

*Dunegan, J. C. & Goldsworthy, M. C. (1948). The control of blossom blight and its relation to brown rot of Red Bird peaches at harvest. *Plant Dis. Reptr*, **32**, 136–137.

Dunlap, A. A. & Langley, B. C. (1941). A cold storage test with peaches sprayed for brown rot. *Progr. Rep. Texas agric. Exp. Sta.*, 761, 1 p.

*Dunn, M. S. (1924). The microloop. A rapid method of isolating single spores. *Phytopathology*, **14**, 338–340.

*Dunn, M. S. (1926). Effects of certain acids and their sodium salts upon the growth of *Sclerotinia cinerea*. *Amer. J. Bot.*, **13**, 40–57.

Dutton, W. C. (1924). Cherry diseases and their control. *Amer. Fruit Gr.*, **44**, pp. 6, 13, 24.

*Dutton, W. C. & Johnston, S. (1922). Dusting and spraying experiments of 1920 and 1921. *Spec. Bull. Mich. agric. Exp. Sta.*, 115, 54 pp.

*EASTHAM, J. W. (1925). Report of Provincial Plant Pathologist, Vancouver. *Rep. B.C. Dep. Agric.* 1924, pp. K32–K34.

*EASTHAM, J. W. (1929). Report of Provincial Plant Pathologist, Vancouver. *Rep. B.C. Dep. Agric.* 1928, pp. 36–40.

*EASTHAM, J. W. (1935*a*). Report of Provincial Plant Pathologist. *Rep. B.C. Dep. Agric.* 1934, pp. R30–R37.

*EASTHAM, J. W. (1935*b*). The relation between climate and the incidence of some orchard diseases in British Columbia. *Proc. 5th Pacif. Sci. Congr.*, 1933, **4**, 3229–3232.

*EHRENBERG, C. G. (1818). *Sylvae mycologicae berolinensis.* Berlin.

*ELSSMANN, E. (1939). Prüfung von Sauerkirschensorten auf ihr Verhalten gegen *Sclerotinia cinerea* Schroet. *Forschungsdienst*, 7, 361–366.

ENGLISH, W. H. (1940). Taxonomic and pathogenicity studies of the fungi which cause decay of pears in Washington. *Res. Stud. State Coll. Wash.*, **8**, 3, 127–128.

*ENGLISH, W. H. (1945). Fungi isolated from mouldy sweet cherries in the Pacific North-West. *Plant Dis. Reptr*, **2**, 559–566.

*ENGLISH, H. & GERHARDT, F. (1942). Effect of carbon dioxide and temperature on the decay of sweet cherries under simulated transit conditions. *Proc. Amer. Soc. hort. Sci.*, **40**, 172–176.

ENGLISH, H. & GERHARDT, F. (1946). The effect of ultraviolet radiation on the viability of fungus spores and on the development of decay in sweet cherries. *Phytopathology*, **36**, 100–111.

*ERIKSSON, J. (1913). Zur Kenntnis der durch Monilia-Pilze hervorgerufenen Blüten- und Zweigdürre unserer Obstbäume. *Mykol. Zbl.*, **2**, 65–78.

*ERIKSSON, J. (1930). *Fungous diseases of plants.* English translation by W. Goodwin, London.

ESBJERG, N. (1929). Forsøg med Rentabiliteten ved Sprøjtning af Aebletraeer. I. *Tidsskr. Planteavl*, **35**, 517–565.

ESBJERG, N. (1938). Forsøg med Opbervaring af Aebler. I. Foreløbig Beretning om Forsøg med forskellige Lagre, uden og med forskellig Nedkøling. *Tidsskr. Planteavl*, **43**, 310–336.

*ESFANDIARI, E. (1947). Diseases of cultivated plants and fruit trees of the sub-tropical regions of northern Iran. *Publ. trimest. Dep. gen. Prot. Pl. Tehran*, No. 5, 1–21.

*ESFANDIARI, E. (1949). Troisième liste des fungi ramassés en Iran. *Ent. Phytopath. appl., Tehran*, 1948, 8, 16 pp.

*ESTIENNE, V. & SOENEN, A. (1946). La moniliose des arbres fruitiers à noyau. *Fruit belge*, **14**, 70–80, 81–95.

EVANS, A. W. & OWENS, C. E. (1941). Incidence of *Sclerotinia fructicola* and *S. laxa* on sweet cherries in Oregon. *Phytopathology*, **31**, 469–471.

*EVREINOFF, V. A. (1943). Le prunier japonais (*Prunus salicina* Lindl.), son origine, ses variétés, sa culture. *Rev. hort. Paris*, **115**, p. 324.

*EZEKIEL, W. N. (1921). Some factors affecting the production of apothecia of *Sclerotinia cinerea*. *Phytopathology*, **11**, 495–499.

EZEKIEL, W. N. (1923). Hydrogen-ion concentration and the development of *Sclerotinia apothecia*. *Science*, **58**, 166.

EZEKIEL, W. N. (1924*a*). Strains of the brown-rot fungus *Sclerotinia americana*. (Abstr.) *Phytopathology*, **14**, 32.

*EZEKIEL, W. N. (1924*b*). Fruit rotting Sclerotinias. II. The American brown-rot fungi. *Bull. Md agric. Exp. Sta.*, 271, 87–142.

EZEKIEL, W. N. (1925*a*). Presence of the European brown-rot fungus in America. (Abstr.) *Phytopathology*, **15**, 55.

EZEKIEL, W. N. (1925*b*). Presence of the European brown-rot fungus in America. *Phytopathology*, **15**, 535–542.

EZEKIEL, W. N. (1926). Fruit-rotting Sclerotinias. II. Longevity of buried brown-rot mummies. *Bull. Md agric. Exp. Sta.*, **284**, 22 pp.

EZEKIEL, W. N. (1927). Two fungi on Sclerotinia apothecia. *Phytopathology*, **17**, 791–792.

*FAES, H. & STAEHELIN, M. (1924). La maladie cryptogamique des Abricotiers en Valais. *Annu. agric. Suisse*, **25**, 22 pp.

*FAES, H. & STAEHELIN, M. (1925*a*). Sur quelques champignons déterminant la pourriture des fruits. *Annu. agric. Suisse*, **26**, 59–72.

*FAES, H. & STAEHELIN, M. (1925*b*). Action cryptogamicide comparée des sels de cuivre, de nickel, de zinc, de fer, et d'aluminium sur divers champignons parasites. *Mém. Soc. vaud. Sci. nat.*, **9**, 73–139.

*FAES, H. & STAEHELIN, M. (1927). Les champignons et les insectes ennemis du cerisier. *Annu. agric. Suisse*, **28**, 1–27.

*FAES, H. & TONDUZ, P. (1923). Station fédérale d'essais viticoles à Lausanne et Domaine de Pully. Rapport Annuel 1922. *Annu. agric. Suisse*, **24**, 26 pp.

*FAES, H. & TONDUZ, P. (1925). Station fédérale d'essais viticoles à Lausanne et Domaine de Pully. Rapport Annuel, 1924. *Annu. agric. Suisse*, **26**, 657–678.

*Faes, H. & Tonduz, P. (1926). Station fédérale d'essais viticoles à Lausanne et Domaine de Pully. Rapport Annuel, 1925. *Annu. agric. Suisse*, **27**, 241–270.

Fant, G. W. (1923*a*). The brown rot canker and twig blight of the peach. *Rep. N.J. agric. Exp. Sta. for the year ending June* 30, 1922, pp. 547–548.

Fant, G. W. (1923*b*). Spray versus dust in controlling the diseases of apples. *Rep. N.J. agric. Exp. Sta. for the year ending June* 30, 1922, pp. 551–553.

Fant, G. W. (1924.) The manner of infection of peach twigs by the brown rot fungus. *Phytopathology*, **14**, 427–429.

Farley, A. J. (1923). Dry-mix sulphur lime. A substitute for self-boiled lime sulphur and summer strength concentrated lime sulphur. *Bull. N. J. agric. Exp. Stas.*, 379, 16 pp.

Farský, O. (1925). [Experiments with some preparations recommended for the protection of plants.] *Ochr. Rost.* **5**, 76–79, 82–89.

*Feekes, F. H. (1949). Onderzoekingen over Schimmelbestrijdingsmiddelen. I. Sporekiemungsproeven met Dithiocarbamaten. *Tijdschr. PlZiekt.*, **55**, 22–30.

*Feichtmeir, E. F. (1949). The effect of particle size and solubility of sulphur in carbon disulfide upon its toxicity to fungi. *Phytopathology*, **39**, 605–615.

*Ferraris, T. (1928*a*). Agricoltura e fitopatologia nel Kashmir. *Curiamo le Piante!* **6**, 81–86. [*R.A.M.* **8**, 89.]

*Ferraris, T. (1928*b*). Mummificazione delle Nespole. *Curiamo le Piante!* **23**, 234–236. [*R.A.M.* **8**, 322].

*Fischer, G. W. (1930). A study of the fruit diseases occurring in a mid-western market. *Butler Univ. bot. Stud.*, **1**, 105–127.

*Fischer, H. (1950*a*). Der Einfluss der Infektion auf die Temperatur und die Atmung pflanzlicher Gewebe. *Phytopath. Z.*, **16**, 171–202.

*Fischer, H. (1950*b*). Kohlensaureabgabe von gesundem und krankem Apfelgewebe und bei *Monilia fructigena* Pers. im Apfelsaft. *Landw. Jb. Schweiz*, **64**, 82–83.

*Fish, S. (1927). Brown rot of peaches. *J. Dep. Agric. Vict.*, **25**, 409–411.

*Fish, S. (1930). Brown rot and transit rot and their control. A report on consignments of peaches sent from Goulburn Valley to the Sydney market. *J. Dep. Agric. Vict.*, **28**, 33–43.

*Fish, S. (1933). Brown rot in peaches. Goulburn valley fruit in Sydney. *J. Dep. Agric. Vict.*, **31**, 381–383; 387.

*Fish, S. (1934). Brown rot control. *Fruit World, Melbourne*, **35**, 367–368.

Fish, S. (1936). Fungus disease control. *Fruit World, Melbourne*, **37**, 5.

*Fisher, D. F. & Brooks, C. (1924). Control of brown-rot of prunes and cherries in the Pacific northwest. *Fmrs' Bull. U.S. Dep. Agric.*, 1410, 12 pp.

Foëx, E. & Chabrolin, C. (1923). Les maladies des arbres fruitiers et leurs traitments. *Lib. Progrés agric. vitic.*, 24 pp., 1923.

*Folke, J. (1950). The effect of several spray materials on the control of blossom blight in sour cherries. (Abstr.) *Phytopathology*, **40**, 967.

*Foote, M. W., Little, J. E. & Sproston, T. J. (1949). On naphthoquinones as inhibitors of spore germination. *J. biol. Chem.*, **181**, 481–487.

*Foster, H. H. (1949). Peach spray experiments for brown rot control in South Carolina during 1948. *Plant Dis. Reptr*, **33**, 471–478.

Foster, H. H. (1952). Organic vs. inorganic fungicides for the control of the brown-rot fungus on peaches. (Abstr.) *Phytopathology*, **42**, 7.

*Frampton, V. L. (1941). Respiration of conidia of *Sclerotinia fructicola*. (Abstr.) *Phytopathology*, **31**, 9.

Frank, B. (1899). Berichtigung zu C. Wehmer, *Monilia fructigena* Pers. *Ber. dtsch. bot. Ges.*, **17**, 40–42.

*Frank, B. & Krüger, F. (1899). Ueber die gegenwärtig herrschende Monilia-Epidemie der Obstbäume. *Landw. Jb.*, **28**, 185–216.

*Fresa, R. (1945). Podredumbre morena de los Durazneros y Ciruelos en el delta del Paraná. *Rev. argent. Agron.*, **12**, 22–25.

Fries, N. (1938). Über die Bedeutung von Wuchsstoffen für das Wachstum veraschiedener Pilze. *Symb. bot. upsaliens.*, **3**, 188 pp. [*R.A.M.*, **18**, 335–336].

*The Fungicide Committee of the American Phytopathological Society (compiled by). (1949) Nationwide results with fungicides in 1948; 4th Annual Report. *Suppl. Plant Dis. Reptr*, 181, 87 pp.

*Gallástegui, I. (1942). [The winter treatment of cider apples.] *Agricultura, Madr.*, **11**, 17–19. [*R.A.M.*, **21**, 338].

*Galloway. (1889). Brown rot of the cherry. *Monilia fructigena* Pers. *Rep. U.S. Dep. Agric., for* 1888, 349–352.

*Garbowski, L. & Juraszkówna (Mme), H. (1933). [Diseases of useful plants in the period 1926 to 1930. A summary of the reports of the Plant Protection Stations.] *Rocznik Ochrony Roślin*, **1**, 165–196. [*R.A.M.*, **13**, 288].

Gard, M. (1931). Sur un traitement du Monilia du prunier d'Ente en 1930. *Rev. path. vég.*, **18**, 112–114.

Gardner, M. W. (1927). Indiana plant diseases, 1925. *Proc. Ind. Acad. Sci.*, **36**, 231–247.

Gardner, M. W. (1928). Indiana plant diseases, 1926. *Proc. Ind. Acad. Sci.*, **37**, 411–426.

*Gaudineau, M. (?1948). Les Monilias des arbres fruitiers et les invasions du *Monilia cinerea*. *C.R. 1er Congr. int. Phytopharm*, 1946, 219–225. [*R.A.M.*, **27**, 429].

Gaudineau (Mlle). (1950). Traitements des arbres fruitiers à réalizer en hiver contre les cryptogames. *I.N.R.A.*, *B.T.I.*, **49**, pp. 1–5.

Gayford, G. W. (1948). Brown rot in stone fruits. *J. Dep. Agric. Vict.*, **46**, 462–463.

Gayford, G. W. (1950). Control fungus diseases in the spring. *J. Dep. Agric. Vict.*, **48**, 367.

*Gerassimenko, L. R. (1926). [Principal insect pests and diseases of cultivated plants in the Ukraine in 1926.] *Protection of plants in Ukraine*, **2**, 7–19. [*R.A.M.*, **6**, 275].

*Gerhardt, F., English, H. & Smith, E. (1945). Cracking and decay of Bing cherries as related to the presence of moisture on the surface of the fruit. *Proc. Amer. Soc. hort. Sci.*, **46**, 191–198.

*Gerhardt, F. & Ryall, A. L. (1939). The storage of sweet cherries as influenced by carbon dioxide and volatile fungicides. *Tech. Bull. U.S. Dep. Agric.*, 631, 20 pp.

Ghillini, C. A. (1951). Danni da Sclerotinia sul l'albicocco. *Not. Mal. Piante*, No. 15, p. 75.

Giddings, N. J. & Wood, J. I. (1925). Diseases of fruit and nut crops in the United States in 1924. *Suppl. Plant Dis. Reptr*, 30, 105 pp.

*Gloyer, W. O. (1928). Report on plant diseases. *Proc. N.Y. St. hort. Soc.*, **70**, 23–29.

*Goldsworthy, M. C. (1944). The fungicidal and phytotoxic behaviour of some selected organic and inorganic copper compounds as related to weathering. (Abstr.) *Phytopathology*, **34**, 991.

*Goldsworthy, M. C., Carter, R. H. & Green, E. L. (1942). The fungicidal and phytocidal properties of some copper xanthates. *Phytopatholoyg*, **32**, 497–504.

*Goldsworthy, M. C. & Gertler, S. I. (1949). Fungicidal and phytotoxic properties of 506 synthetic organic compounds. *Suppl. Plant Dis. Reptr*, **182**, 89–109.

*Goldsworthy, M. C. & Gertler, S.I. (1951). Fungicidal and phytotoxic properties of 412 synthetic organic compounds. *Suppl. Plant Dis. Reptr*, **205**, 176–189.

*Goldsworthy, M. C. & Green, E. L. (1933). Some promising fungicides. *Phytopathology*, **23**, 561–562.

*Goldsworthy, M. C. & Green, E. L. (1936). Availability of the copper of Bordeaux mixture residues and its absorption by the conidia of *Sclerotinia fructicola*. *J. agric Res.*, **52**, 517–533.

*Goldsworthy, M. C. & Green, E. L. (1938). Effect of low concentrations of copper on germination and growth of conidia of *Sclerotinia fructicola* and *Glomerella cingulata*. *J. agric. Res.*, **56**, 489–505.

*Goldsworthy, M. C. & Green, E. L. (1939). The fungicidal activity of phenothiazine and some of its oxidation derivatives. *Phytopathology*, **29**, 700–716.

*Goldsworthy, M. C., Green, E. L. & Haller, H. L. (1942). Fungicidal properties of 2,4-diaminodiphenylamine and other substituted diphenylamines. *J. agric. Res.*, **64**. 667–678.

*Gonçalves, R. D. (1939). [Scab and brown rot of the peach.] *Biologico*, **5**, 17–18.

*Goossens, J. (1938). Monilia-ziekten van appel, peer, kers, morel, pruim, abrikoos en perzik. Meded. *PlZiektDienst*, *Wageningen*, **88**, 5 pp.

*Gortner, R. A. (1920). The biochemistry of the resistance to disease in plants. *Minn. St. Rep.*, 1920, 27–28.

Gottlieb, D. (1945). A comparison of the agar plate and test-tube-dilution methods for the preliminary evaluation of fungicides. (Abstr.) *Phytopathology*, **35**, 485.

*Gottlieb, D., Bhattacharyya, P. K., Carter, H. E. & Anderson, H. W. (1951). Endomycin, a new antibiotic. *Phytopathology*, **41**, 393–399.

*Gottlieb, D., Hassan, H. H. & Linn, M. B. (1950). Actidione as a plant protectant. *Phytopathology*, **40**, 218–219.

Gram, E. & Stapel, C. (1935). Forsøg med Foraarskarbolineum. *Medd. Forsøgsv. Plantek. Kbh.*, **242**, 3 pp.

*Gram, E. & Weber, Anna. (1940). *Plantesygdomme*, pp. 119–120, 173–176. Copenhagen.

Green, D. E. (1929). Brown rot of apples. *Gdnrs' Chron.*, **86**, 507.

*Gries, G. A. (1943). Juglone (5-hydroxy-1, 4-naphthoquinone)—a promising fungicide. (Abstr.) *Phytopathology*, **33**, 1112.

Groves, A. B. (1944). Sulphur sprays. *Bull. Va agric. Exp. Sta.*, 359, 22 pp.

*Grubb, N. H. (1921). Tests of fungicides on apple trees carried out at East Malling Research Station. *J. Pomol.*, **2**, 93–114.

Guyot, A. L. (1928). Considérations générales sur l'état sanitaire des plantations fruitières de la Vallée du Rhone. *Rev. Path. vég.*, **25**, 210–224.

Guyot, A. L. (1929). Les maladies des arbres fruitiers à noyau. *Librarie Prog. agric.*

Guyot, L. & Joëssel, H. (1926). Contribution à l'étude des traitements rationnels des vergers. *Rev. Path. vég.*, **13**, 219–240.

Hadorn, C. (1941). Spätfröste, verregnete Blütezeit, Monilia Blüten- und Zweigdürre an Apfelbaumen. *Schweiz. Z. Obst- u. Weinb.*, **50**, 347–350.

*Hafiz, A. (1946). Brown rot of stone and pome fruits. *Punjab Fruit J.*, **10**, 145–146.

*Hall, Muriel P. (1933). An analysis of the factors controlling the growth form of certain fungi, with special reference to *Sclerotinia* (*Monilia*) *fructigena*. *Ann. Bot., Lond.*, **47**, 543–578

*Hallemans, A. (1946) De Moniliaziekte en haar mogelijke bestrijding. *Cult. Hand.*, 1946, pp. 17–18.

Haller, M. H., Smith, W. L., Jr., Smith, M. A., Womeldorph, S. E., Jr., & Wright, W. R. (1952). Postharvest fungicidal treatments of peaches tested in 1951. (Abstr.) *Phytopathology*, **42**, 341–342.

*Hamilton, J. M. (1931). Studies of the fungicidal action of certain dusts and sprays in the control of apple scab. *Phytopathology*, **21**, 445–523.

*Hamilton, J. M. (1948). Peach leaf curl and brown-rot control. *Rep. N.Y. St. agric. Exp. Sta.*, 1947, 30–31.

*Hamilton, J. M., Palmiter, D. H. & Mack, G. L. (1943). Particle size of sulphur and copper fungicides in relation to apple scab and cedar apple rust control. *Phytopathology*, **33**, 533–550.

*Hamilton, J. M., Palmiter, D. H. & Mack, G. L. (1944). Tests with new organic fungicides on orchard fruits. (Abstr.) *Phytopathology*, **34**, 1002.

*Hamilton, J. M. & Palmiter, D. H. (1951). Orchard tests for apple scab control in New York State: I. Sulfur fungicides. *Bull. N.Y. St. agric. Exp. Sta.*, 747, 63 pp.

Hamilton, J. M. & Weaver, L. O. (1943). Freezing preservation of fungi and fungus spores. *Phytopathology*, **33**, 612–613.

*Hammond, A. A. (1922). Spraying experiments for brown rot of stone fruit (*Sclerotinia fructigena*). *J. Dep. Agric. Vict.*, **20**, 182–189.

*Hammond, A. A. (1923). Brown rot of stone fruit. Spraying experiments. *J. Dep. Agric. Vict.*, **21**, 489–493.

Hamner, C. L., Sell, H. M., Klomparens, W. & Vaughn, J. R. (1950). Selective inhibition of the growth of green plants and fungi by beta methyl umbelliferone. *Bot. Gaz.*, **112**, 135–137.

*Hansen, H. N. & Thomas, H. E. (1940). Flower blight of Camellias. *Phytopathology*, **30**, 166–170.

Hansen, H. R. & Weber, Anna. (1936). Smitsomme sygdomme af saerlig interesse. *Tidsskr. Planteavl*, **41**, 557–562.

*Hansen, H. R. & Weber, Anna. (1950). [Plant diseases and pests in Denmark, 1948.] *Tidsskr. Planteavl*, **54**, 1–61.

*Harrison, T. H. (1922). Note on the occurrence in New South Wales, Australia, of the perfect stage of a Sclerotinia causing brown rot of fruits. *J. roy. Soc. N.S.W.*, **55**, 215–219.

*Harrison, T. H. (1928). Brown rot of fruits, and associated diseases in Australia. Part I. History of the disease and determination of the causal organism. *J. roy. Soc. N.S.W.*, **62**, 99–151.

*Harrison, T. H. (1933). Brown rot of fruits and associated diseases of deciduous fruit trees. I. Historical review, and critical remarks concerning taxonomy and nomenclature of the causal organisms. *J. roy. Soc. N.S.W.*, **67**, 132–177.

*Harrison, T. H. (1935*a*). Brown rot of fruits and associated diseases of deciduous fruit trees. II. The apothecia of the causal organisms. *J. roy. Soc. N.S.W.*, **68**, 154–176.

*Harrison, T. H. (1935*b*). Brown rot of fruits and associated diseases in Australia. II. An interesting Discomycete, *Sclerotinia aestivalis* Pollock, occurring on mummified fruits. *Mycologia*, **27**, 302–318.

Harrison, T. H. (1940). Proceedings: Discussion on plant diseases and the weather. *Trans. Brit. mycol. Soc.*, **24**, 264–266.

*Harrison, T. H. & El-Helaly, A. F. (1935). On *Lambertella corni-maris* von Höhnel, a brown-spored parasitic discomycete. *Trans. Brit. mycol. Soc.*, **19**, 199–214.

*Hart, R. (1947). The new Merton cherries. *J. Pomol.*, **23**, 112–116.

Hartley, C. (1924). De ziekten van de cacao. *Bull. Inst. PlZiekt.*, 19, 16 pp. [*R.A.M.* **4**, 149.]

Harvey, J. M. & Hansen, H. N. (1950). Camellia flower blight and its control. (Abstr.) *Phytopathology*, **40**, 965–966.

*Hawker, Lilian E. (1950). *Physiology of Fungi*, 359 pp. London.

*Hawkins, L. A. (1915). Some effects of the brown-rot fungus upon the composition of the peach. *Amer. J. Bot.*, **2**, 71–81.

Headlee, T. J., Martin, W. H. & Farley, A. J. (1930). 1930 spray schedule for plums and cherries. *Circ. N.J. agric. Exp. Sta.*, 221, 4 pp.

Headlee, T. J., Martin, W. H. & Farley, A. J. (1930). 1930 spray schedule for peaches. *Circ. N.J. agric. Exp. Sta.*, 222, 4 pp.

Heald, F. D. (1915). Brown rot of stone fruits. *Wash. Agric.* **8**, 6 pp.

*Heiberg, Barbara C. & Ramsey, G. B. (1946). Fungistatic action of diphenyl on some fruit and vegetable pathogens. *Phytopathology*, **36**, 887–891.

*Henning, E. (1922). Avdelning för landtbruks botanik. *K. LandtbrAkad. Handl. Stockh.*, 1922, 26–32. [*R.A.M.*, **1**, 417–418.]

*Henrick, J. O. (1935). Brown rot of stone fruits, *Sclerotinia cinerea* (Bon.) Schroet., syn.: *Monilia cinerea* (Bon.). *Tasm. J. Agric.*, **6**, 73–79.

Hesler, L. R. (1926). Peach disease conditions in Ohio. *Bimon. Bull. Ohio agric. Exp. Sta.*, 11, 110–114.

*Hesse, C. O. (1939). Variation in resistance to brown rot in apricot varieties and seedling progenies. *Proc. Amer. Soc. hort. Sci.*, **36**, 266–268.

*Hessler, G. (1942). Controlling brown rot on ripening peaches. *Ann. Rep. Oregon St. hort. Soc.*, **33**, 58–61.

*Heuberger, J. W. (1934). Fruit-rotting Sclerotinias. IV. A cytological study of *Sclerotinia fructicola* (Wint.) Rehm. *Bull. Md agric. Exp. Sta.*, 371, 167–189.

*Heuberger, J. W., Munger, G. D. & Poulos, P. L. (1949). Post-harvest chemical dip treatment of peaches for control of brown-rot disease in the package. (Abstr.) *Phytopathology*, **39**, 9.

*Heuberger, J. W., Poulos, P. L. & Hood, J. J. (1950). Peach brown rot: liquid lime sulphur and certain chlorine compounds as post-harvest dip treatments for control in the package. (Abstr.) *Phytopatholoyg*, **40**, 12.

*Hewitt, W. B. & Leach, L. D. (1939). Brown rot sclerotinias occurring in California and their distribution on stone fruits. *Phytopathology*, **29**, 337–351.

Heydemann, F. (1928). Zur Bekämpfung der Moniliakrankheit an Sauerkirschen. *Obst- und Gemüsebau*, **74**, 170–171.

Hienton, T. E. & Fawcett, K. I. (1939). Precooling tests of Indiana strawberries, cantaloupes and peaches. *Bull. Ind. agric. Exp. Sta.*, 439, 36 pp.

Hildebrand, E. M. (1939). Two fungi (*Valsa leucostoma* and *V. cincta*) besides brown rot (*Sclerotinia fructicola*) prominently involved in peach-canker complex. (Abstr.) *Phytopathology*, **29**, 10–11.

Hildebrand, E. M. (1947). Perennial peach canker and the canker complex in New York with methods of control. *Mem. Cornell agric. Exp. Sta.*, 276, 61 pp.

Hino, I. (1929). Microconidia in genus *Sclerotinia* with special reference to the conidial forms in the genus. *Bull. Miyazaki Coll. Agric.*, **1**, 67–90.

*Hoerner, G. R. (1930). Dusting in the Northwest. *Bett. Fruit*, **24**, 9–10.

*von Höhnel, F. (1918). *Lambertella*. *Fragm.* 2 *Mykol.*, **21**, No. 1078, 47.

*Honey, E. E. (1928). The Monilioid species of *Sclerotinia*. *Mycologia*, **20**, 127–157.

*Honey, E. E. (1936). North American species of *Monilinia*. I. Occurrence, grouping, and life-histories. *Amer. J. Bot.*, **23**, 100–106.

*Honey, E. E. (1940). *Monilinia* causing a brown rot and blight of the common azalea. *Phytopathology*, **30**, 537–538.

*Honey, E. E. (1942). *Monilinia amelanchieris*. *Mycologia*, **34**, 575–578.

Hood, J. J. & Heuberger, J. W. (1950). Peach brown rot: Effect of low temperature storage for various periods on subsequent rot development in the package. (Abstr.) *Phytopathology*, **40**, 13.

*Hopperstead, S. L., Manns, T. F. & Heuberger, J. W. (1945). Department of Plant Pathology. *Rep. Del. agric. Exp. Sta. for* 1944–45, 33–39.

*Horne, A. S. (1933). Biological work on fruit. *Rep. Food Invest. Bd, Lond.*, 1932, pp. 279–300.

*Horne, A. S. (1934). Biological work on fruit. *Rep. Food Invest. Bd, Lond.*, 1933, pp. 228–245.

*Horsfall, J. G. (1945). *Fungicides and their action*, 239 pp. Waltham, Mass.

*Horsfall, J. G. (1951). Relative specificity of *Sclerotinia* and *Stemphylium* to toxicants. (Abstr.) *Phytopathology*, **41**, 658.

*Horsfall, J. G. & Rich, S. (1951). Fungitoxicity of heterocyclic nitrogen compounds. *Contr. Boyce Thompson Inst.*, **16**, 313–347.

Houben, J. & Wollenweber, H. W. (1929). Hexylresorcin und Phenyläthyl gegen pflanzenpathogene Pilze. *Biochem. Z.*, **204**, 448–455.

Howard, F. L. (1940). The value of testing fungicides in the laboratory before use in the field. *Proc. Amer. Soc. hort. Sci.*, **37**, 409–414.

Howard, W. L. (1920). Brown rot of apricots and its prevention. *Bett. Fruit*, **15**, p. 7.

*Howard, W. L. (1922). The apricot in California. *Circ. Univ. Calif.*, 238, 53 pp.

*Howard, W. L. & Horne, W. T. (1921). Brown rot of apricots. *Bull. Calif. agric. Exp. Sta.*, 326, 73–88.

Huber, G. A. & Baur, K. E. (1938). Progress report of brown rot investigation conducted during 1937 in Clark County, Washington. *Plant Dis. Reptr*, **22**, 14–15.

*Huber, G. A. & Baur, K. (1939*a*). The use of calcium cyanamid for the destruction of apothecia of *Sclerotinia fructicola*. *Phytopathology*, **29**, 436–441.

Huber, G. A. & Baur, K. E. (1939*b*). The occurrence of *Sclerotinia fructicola* and *S. laxa* on stone fruits in Western Washington. (Abstr.) *Phytopathology*, **29**, 825.

Huber, G. A. & Baur, K. (1940). Department of Plant Pathology. *Rep. Wash. agric. Exp. Sta.*, pp. 37–42.

*Huber, G. A. & Baur, K. (1941). Brown rot on stone fruits in Western Washington. *Phytopathology*, **31**, 718–731.

*Huber, G. A. & Baur, K. (1942). Apothecia of *Sclerotinia fructicola* on peach in Western Washington. *Phytopathology*, **32**, 635–636.

Huber, G. A., Baur, K. E. & Breakey, E. P. (1939). The effect of calcium cyanamid on development of apothecia of *Sclerotinia fructicola* and on population of *Taeniothrips inconsequens* in prune orchards. (Abstr.) *Phytopathology*, **29**, 825.

Humphrey, J. E. (1891). The brown rot of stone fruits (*Monilia fructigena* Pers.). *Ann. Rep. Mass. agric. Exp. Sta.* 1890, p. 213.

*Humphrey, J. E. (1893). On *Monilia fructigena*. *Bot. Gaz.*, **18**, 85–93.

*Hunt, T. F. (1923). Brown rot of apricots. *Rep. Calif. Coll. Agric.* 1922/23, p. 183.

*Hurt, R. H. (1935). Spraying and dusting peaches for brown rot control. *Amer. Fruit Gr.*, **55**, pp. 7, 13.

*Hurt, R. H. (1937). 1. The control of peach leaf curl, scab and brown rot. 2. Spray materials for peaches. *Bull, Va Truck Exp. Sta.* 312, 16 pp.

Hurt, R. H. (1939). Bordeaux mixture as a summer fungicide for peaches. (Abstr.) *Phytopathology*, **29**, 11.

Hurt, R. H. & Schneiderhan, F. J. (1929). Calcium sulphide for the control of apple and peach diseases. *Tech. Bull. Va agric. Exp. Sta.* 36, 15 pp.

*Hus, P. (1933). De ontwikkeling van carbolineum als bestrijdingsmiddel in de fruitteelt. *Tijdschr. PlZiekt.*, **39**, 232–245.

*Husz, B. (1936). [Spray experiments for the control of Monilia blight of apricots and sour cherries, 1931–1936]. *Bull. R. Hungarian hort. Coll.*, **2**, 14–31.

*Hutton, K. E. (1947*a*). Diseases of cherries. Brown rot. *Agric. Gaz. N.S.W.*, **58**, 369–370.

*Hutton, K. E. (1947*b*). Brown rot of stone fruits. *Agric. Gaz. N.S.W.*, **58**, 487–491.

Hyde, W. C. (1919). Control of brown-rot of stone-fruits. The Stoke experiments: season 1918–19. *N.Z.J. Agric.* **19**, 24–25.

*Ingram, C. (1948). *Ornamental cherries*, 259 pp. London.

*Istvánffi, G. (1906). Adatok a gyümölesfák Monilia betegségének ismeretśkez. *A.M.K. Közp. Szölész. Kíserl. Áll. és Ampeologiai Intézet Évkönyve* 1 évf., 83–97. [Quoted by Husz, 1936.]

*Jaap, O. (1908). Beiträge zur Pilzflora der österreichischen Alpenländer. I. Pilze aus Südtirol und Kärnten. *Ann. mycol., Berl.*, **6**, 192–221.

*Jackson, H. S. (1915*a*). Pear canker. *Monilia* sp. *Bienn. Crop. Pest Rep. Ore.*, **2**, 271–272.

Jackson, H. S. (1915*b*). Prune brown rot on dried fruit. *Sclerotinia fructigena* (Pers.) Schroet. *Bienn. Crop Pest Rep. Ore.*, **2**, 276–277.

*Janke, A. & Beran, F. (1933). Über die mikrobicide Wirkung von organischen Säuren und ihren Kupfersalzen. Ein Beitrag zum Problem des Zusammenhangs zwischen chemischer Konstitution und mikrobicider Wirkung. *Arch. Mikrobiol.* **4**, 54–71. [*R.A.M.* **12**, 523].

Jehl, R. A. (1914). Peach cankers and their treatment. *Circ. Cornell agric. Exp. Sta.*, 26, pp. 53–64.

*Jirak, L. (1942). Über die enzymatischen Vorgänge des Welkens bei jungen belaubten Marillenbäumen. *Gartenbauwiss.* **17**, 18.

*Joëssel, P. H. (1928*a*). Le Monilia de l'Abricotier dans la moyenne Vallée du Rhône et en Provence. *Rev. Path. vég.*, **15**, 198–209.

*Joëssel, P. H. (1928*b*). Quelques maladies du Pêcher et de l'Abricotier dans la region rhodanienne. *Prog. agric.*, **90**, 350–353, 370–374.

*Joëssel, P. H. (1929). Quelques considerations sur les traitements des arbres fruitiers dans la basse vallée du Rhône. *Rev. Path. vég.*, **16**, 71–88.

*Joëssel, P. H. (1932*a*). Le problème du Monilia de l'abricotier dans la vallée du Rhône. *Bull. Off. rég. agric. du Midi*, 44, 69 pp.

*Joëssel, P. H. (1932*b*). Quelques observations sur les traitements à envisager pour l'abricotier et le pêcher dans la vallée du Rhône. *Rev. Path. vég.*, 88–105.

Joëssel, P. H. (1936). Les principales maladies cryptogamiques des arbres fruitiers en Provence. *Fruits Primeurs*, **6**, 346–350.

Joëssel, P. H. (1937). Les principales maladies cryptogamiques des arbres fruitiers en Provence. *Fruits Primeurs*, **7**, 49–54.

*Joëssel, P. H. & Anrès, M. (1932). Résultats des essais de traitement contre le Coryneum de l'Abricotier dans la région de Barroux (Vaucluse) au cours des années 1931 et 1932. *Rev. Path. vég.*, **19**, 253–255.

Joëssel, P. H. & Bordas, J. (1931). Recherches sur les dépérissements de l'abricotier dans la vallée du Rhône (1927 à 1930). *Ann. Epiphyt.*, **8**, 359.

*Johansen, G. (1937). Smitsomme Sygdomme af saerlig Interesse. *Tidsskr. Planteavl*, **42**, 229–230.

Johansen, G. (1945). *Monilinia fructigena* (Aderh. & Ruhl.) Honey i Danmark. *Friesia*, **3**, 111–114.

*Johansen, G., Hansen, H. R., Nielsen, O. & Weber, Anna. (1937). Undersøgelser over *Monilia fructigenas* smitteevne. *Tidsskr. Planteavl*, **42**, 229–230.

*Johnson, F. (1950). The effect of several spray materials on the control of blossom blight in sour cherries. (Abstr.) *Phytopathology*, **40**, 967.

Johnson, J. & Hoggan, I. A. (1937). The inactivation of the ordinary tobacco-mosaic virus by microorganisms. *Phytopathology*, **27**, 1014–1027.

*de Jubainville, d'Arbois, A. & Vesque, J. (1878). *Les maladies des plants cultivées des arbres fruitiers et forestiers*. Paris.

Jørstad, I. (1922). Oversikt over forsoksspøritninger mot soppsykdommer i frukthaven i året 1921. *Norsk Havetidende*, **38**, 4, pp. 75–81; 5, 108–112. [*R.A.M.*, **1**, 389.]

*Jørstad, I. (1923). *Beretning om plantesykdommer i land og havebruket* 1920–22. *II. Frukttraer og baervekster*, 73 pp. Christiana.

*Jørstad, I. (1932). Nyere resultater av forsøkssprøitning mot soppsykdommer på frukttraer. *Medlemsskr. Havedyrkn. Venners*, 2, 7 pp.

Jørstad, I. (1937). Melding om plantesykdommer i land- og hagebruket. Sprøiteforsøk mot soppsykdommer på frukttraer, 61 pp. Oslo.

*Jørstad, I. (1938). Monilia på steinfrukt. *Medlemsskr. Selsk. Havedyrkn. Venners*, 16, 19–20.

*Jørstad, I. (1945). Parasittsoppene på kultur- og nyttevekster i Norge. I. Sekksporesopper (Ascomycetes) og konidiesopper (Fungi imperfecti). *Meld. Sta. plpat. Inst.*, **1**, 69–71.

Kadow, K. J. & Anderson, H. W. (1935). The role of zinc sulphate in peach sprays. *Bull. Ill. agric. Exp. Sta.*, 414, 207–255.

Kadow, K. J. & Anderson, H. W. (1936). Further studies on zinc sulphate in peach sprays with limited tests in apple sprays. *Bull. Ill. agric. Exp. Sta.*, 424, 131–144.

*Karel, G. (1949). Malatya Kayisilarinda Monilia hastaliği. *Mahsul Hekimi* (*Plant Prot. J.*), **2**, 179–182. [*R.A.M.*, **30**, 171.]

*Karsner, H. T. & Saphir, O. (1926). Influence of high partial pressures of oxygen on the growth of certain molds. *J. infect. Dis.*, **39**, 231–236.

*Katser, Annie. (1933). Ueber die Resistenz verschiedener Apfelsorten gegenüber *Sclerotinia fructigena* (Pers.) Schroet. und ihre Beziehung zur Wasserstoffionenkonzentration. *Phytopath. Z.*, **6**, 177–227.

*Kazanskiĭ, A. N. (1935). [The importance of *Rhynchites bacchus* L. in spreading Brown Rot of fruit.] *Bull. Pl. Prot., Leningr.*, 10, 171 pp. [*R.A.E.* (A), **25**, 505–506.]

*Keil, J. (1950). Neue Bakterien mit vornehmlich fungostatischen Stoffwechselprodukten. *Naturwissenschaften*, **37**, 476–477. [*R.A.M.*, **30**, 237–238.]

Keitt, G. W. (1935). Progress in the development of eradicant fungicides. *Phytopathology*, **25**, 23.

*Keitt, G. W., Moore, J. D., Calavan, E. C. & Shay, J. R. (1943). Occurrence of the imperfect stage of *Sclerotinia laxa* on *Prunus cerasus* in Wisconsin. *Phytopathology*, **33**, 1212–1213.

*Keitt, G. W. & Palmiter, D. H. (1937). Potentialities of eradicant fungicides for combating apple scab and some other plant diseases. *J. agric. Res.*, **55**, 397–437.

Kephart, J. E. & Dunegan, J. C. (1949). Studies on the overwintering of *Monilinia fructicola* in peach twig cankers. (Abstr.) *Phytopathology*, **39**, 503–504.

Kidd, M. H. & Beaumont, A. (1924). Apple rot fungi in storage. *Trans. Brit. mycol. Soc.*, **10**, 98–118.

*Kidd, M. N. & Tomkins, R. G. (1928). Fungal diseases of imported fruits. *Rep. Food Invest. Bd, Lond*, 1927, 45–48.

Killian, C. (1919). Sur les causes de la specialisation des Ascomycetes. Le *Monilia cinerea* de cerises. *Ann. Épiphyt.*, **6**, 331–333.

*Killian, C. (1926). Variations des caractères morphologiques et biologiques chez les Ascomycètes et les Deutéromycètes parasites. *Rev. Path. vég.*, **13**, 129–166.

*Killian, K. (1917). Über die Unterschiede der *Monilia cinerea* an Süss- und Sauerkirschen. *Jber. Ver. angew. Bot.*, **15**, 158–160.

*Killian, K. (1921). Über die Ursachen der Spezialisierung bei den Askomyzeten. I. Die *Monilia cinerea* der Kirschen. *Zbl. Bakt.*, **53**, 560–597.

*Kirk, T. W. (1905). Brown-rot (*Sclerotinia fructigena*). *Rep. N.Z. Dep. Agric.*, 1905, p. 425.

*Kirtland, J. R. (1885). Premature decay of the plum. *Florist and Hort. J.*, **4**, 34–36.

*Kivilaan, A. (1936). [Observations on the diseases and injuries of fruit-trees in Estonia in summer of 1935]. *Bull. phytopath. Exp. Sta. Univ. Tartu, Estonia*, **33**, 8 pp.

Klebahn, H. (1930). Die Monilia-Krankheit der Kirschen. *Kranke Pflanze*, **7**, 37–40.

Klemm, M. (1941). Pflanzenschutz in der UdSSR. *Angew. Bot.*, **23**, 41–62.

*Knoppien, P. (1949). Het scheuren van kersen. *Meded. Direct. Tuinb.*, **12**, 77–78.

*Köck, G. (1910). Beobachtungen über den Befall verschiedener Kirschen-und Weichselsorten durch den Moniliapilz. (*Sclerotinia cinerea* [Bon.] Schröt.) *Z. landw. VersWes. Ost.*, **13**, 889–890.

*Kögl, F. & Fries, N. (1937). Über den Einfluss von Biotin, Aneurin und Meso-Inosit auf das Wachstum verschiedener Pilzarten. 26 Mitteilung über pflanzliche Wachstumstoffe. *Hoppe-Seyl. Z.*, **249**, 93–110. [*R.A.M.*, **17**, 196.]

*Köhler, H. (1950). Antibiotika und ihre Bedeutung in der Pflanzenpathologie. *NachrBl. dtsch. PflSchDienst*, **4**, 161–164; 185–193.

*Kotte, W. (1948). *Krankheiten und Schädlinge im Obstbau*, p. 31. Berlin.

*Kovalev, —. (1940). [Immunity of fruit trees to fungus diseases.] *C. R. Acad. Sci. U.R.S.S.*, **27**, 176–179.

Krüger, —. (1905). Praktische Bekämpfung der wichtigsten Obstschädlinge. *Vortrag Obstbau-Vortrags-Kursus LandwKammer Prov. Brandenburg*, Berlin, 14 pp.

*Kunze, G. & Schmidt, J. C. (1817). *Mycologische Hefte* I: 80.

Laibach, F. (1928). Über Zellfusionen bei Pilzen. *Planta*, **5**, 340–357.

*Laidlaw, W. & Brittlebank, C. C. (1922). Brown rot of stone fruits. *J. Dep. Agric. Vict.*, **22**, 442–443.

Lapédagne. H. (1946). Les cognassiers disseminateurs de la moniliose. *Rev. hort. Paris*, **30**, 3, 44–45.

Laubert, R. (1923). Ueber besonders heftiges Auftreten einiger Frühjahrs-krankheiten von Ziergehölzen im Jahre 1923. *Gartenwelt*, **27**, 222–224.

Laubert, R. (1944). Lagerfäulen des Obstes. *Kranke Pflanze*, **21**, 2–4.

Leach, L. D. & Hewitt, W. B. (1939). Forced ejection of ascospores from apothecia of Sclerotinia species. *Phytopathology*, **29**, 373.

Lebedeva, L. A. (1926). [Black rot of fruit trees.] *Defense Plants, Leningrad*, **2**, 588–592.

*Leben, C. & Keitt, G. W. (1947). The effect of an antibiotic substance on apple leaf infection by *Venturia inaequalis*. (Abstr.) *Phytopathology*, **37**, 14.

*Leben, C. & Keitt, G. W. (1948). An antibiotic substance active against certain phytopathogens. *Phytopathology*, **38**, 899–906.

*Lees, A. H. & Briton-Jones, H. R. (1925). Plum aphis and brown rot control. *J. Pomol.*, **4**, 196–199.

Leishman, E. (1939). Brown rot (*Sclerotinia fructicola*) of stone fruit. *J. Dep. Agric. S. Aust.*, **43**, 196–201.

*Leszczenko, P. (1928). [Studies on the action of solutions of salts, alkalis and acids on the spores of some pathogenic fungi.] *Prace Wydz. Chorób Roślin w Bydgoszczy Pánstw. Inst. Nauk. Gospod. Wiejsk.*, **6**, 37 pp. [*R.A.M.*, **8**, 392].

*Lewis, F. H. (1950). Some recent work on peach brown rot and bacterial spot. *State hort. Ass. Penn. News*, **27**, 80–86.

*Liming, O. N. (1932). The relation of pentathionic acid and its component constituents to the toxicity of sulphur fungicides. *Phytopathology*, **22**, 143–165.

*Liming, O. N. (1933). The preparation and properties of pentathionic acid and its salts; its toxicity to fungi, bacteria and insects. *Phytopathology*, **23**, 155–174.

*Liming, O. N. & Young, H. C. (1930). Toxicity of sulphur to spores of *Sclerotinia cinerea* as affected by the presence of pentathionic and other sulphur acids. *J. agric. Res.*, **40**, 951–962.

*Lin, Ch'wan-Kwang. (1940). Germination of the conidia of *Sclerotinia fructicola*, with special reference to the toxicity of copper. *Mem. Cornell agric. Exp. Sta.*, **233**, 33 pp.

*Lin, Kung-Hsiang. (1942). A physiological study of the susceptibility of the blushed and green sides of apples to certain fungous rots. *Phytopathology*, **32**, 239–245.

*Lin, Kung-Hsiang. (1948*a*). The effect of modified air on the rotting of apples in storage. *Lingnan Sci. J.*, **22**, 133–138.

*Lin, Kung-Hsiang. (1948*b*). Enzyme and toxic substance production by apple rotting fungi. *Lingnan Sci. J.*, **22**, 139–142.

*Lindfors, T. & Holmberg, Ch. (1941). Växtsjukdomar i Sverige, 1933–1937, *Medd. Växtskyddanst. Stockh.* **33**, 93–97.

*Ling, L. (1948). Host index of the parasitic fungi of Szechwan, China. *Suppl. Plant Dis. Reptr*, **173**, 16, 29.

*Link, G. K. K. & Wilcox, Hazel W. (1933). Precipitin-ring test applied to fungi. II. *Bot. Gaz.*, **95**, 1–34.

*Linsbauer, L. (1925). Botanisches Versuchslaboratorium und Laboratorium für Pflanzenkrankheiten. *Ber. Bundes-Lehr- u. Versuchstanst. Klosterneuburg*, 1919–20—1924–25, 26–42. [*R.A.M.*, **6**, 337].

*Little, J. E., Sproston, T. J. & Foote, M. W. (1948). Isolation and antifungal action of naturally occurring 2-methoxy-1, 4-naphthoquinone. *J. biol. Chem.*, **174**, 335–342.

*Little, J. E. Sproston, T. J. & Foote, M. W. (1949). Synthesis and antifungal action of 2-methylmercapto-1, 4-naphthoquinone. *J. Amer. chem. Soc.*, **71**, 1124.

*Lopatin, M. I. (1936). [Blossom blight of apples caused by *Sclerotinia fructigena* Schröt.]. *Pl. Prot. Leningr.*, **10**, 103–106.

LOUKYANOVITCH, F. K., LEBEDEVA, L. A., KIZERITZKY, V. A., ERMOLAYEVA O. I. & OBOLENSKY, S. I. (1931). [Pests and diseases of agricultural plants in the region of the Turkestan–Siberian railway]. *Pl. Prot. Leningr.*, **7**, 349–360. [*R.A.M.*, **11**, 316].

*LUCAS, E. H., LEWIS, R. W. & SELL, H. M. (1946). An antibiotic principle derived from seeds of *Brassica oleracea*. *Quart. Bull. Mich. agric. Exp. Sta.*, **29**, 4–6.

LUDWIGS, K. (1923). Bericht über das Auftreten der Spitzendürre (Monilia) bei Kirschen in der Provinz Brandenburg im Jahre, 1922. *Dtsch. ObstZtg.*, **49**, 91–92.

LUDWIGS, K. (1926). Beobachtung über Krankheiterscheinungen am Obst. *Obst- u. Gemüseb.*, **72**, 265–266.

*LUDWIGS, K. (1928). Spirtzkalender für die Bekämpfung der wichtigsten Schädlinge und Krankheiten der Kirschbäume. *Obst- u. Gemüseb.*, **74**, 58.

LYCKÅS, C. (1941). Skador på lagrad frukt. *Fruktodlaren*, **3.**

*MACHACEK, J. E. (1928). Studies on the association of certain phytopathogens. *Rep. Quebec Soc. Prot. Pl.*, 1927–1928, pp. 16–63.

*MADER, E. O. & TELLER, M. N. (1947). Differentiation of cultural types of *Sclerotinia* spp. by means of hydrogen-ion concentration. (Abstr.) *Phytopathology*, **37**, 15.

*MAIER, W. (1942*a*). Ueber ein Zweigsterben der Aprikosen als Folge von Monilia Fruchtfäule. *Z. PflKrankh.*, **52**, 91–107.

*MAIER, W. (1942*b*). Ausmass, Ursache und Verhütung der Monilia-Fäule bei Aprikosen. *Angew. Bot.*, **24**, 303–321.

*MAIER, W. & MITTMANN-MAIER, GERTRUD. (1940). *Monilia cinerea* Bon. als Erreger einer Blattkrankheit an Süsskirsche. *Angew. Bot.*, **22**, 79–85.

*MALENÇON, G. & DELÉCLUSE, R. (1937). Champignons pathogènes observés au Maroc. *Bull. Soc. Sci. nat. Maroc.*, **17**, 132–144.

*MALKOFF, K. (1908). Erster Beitrag zur Kenntnis der Pilzflora Bulgariens. *Ann. mycol. Berl.*, **6**, 29–36.

*MANARESI, A. (1920). Alcune osservazioni sulla *Monilia* del melo. *Riv. Patol. veg.*, **10**, 14 pp.

*MANLEY, H. (1947). The use of iodized fruit wrappings. *Brit. Packer*, **9**, pp. 2–3, 15.

*MARCHALL, EL. & MARCHALL, EM. (1921). Contribution à l'étude des champignons fructicoles de Belgique. *Bull. Soc. Bot. Belg.*, 54, 31 pp.

*MARSH, P. B. (1945). Salts as antidotes to copper in its toxicity to the conidia of *Sclerotinia fructicola*. *Phytopathology*, **35**, 54–61.

*MARSH, R. W. (1929). Investigations on the fungicidal action of sulphur. III. Studies on the toxicity of sulpheretted hydrogen and on the interaction of sulphur with fungi. *J. Pomol.*, **7**, 237–250.

*MARSH, R. W. (1947). Fruit spraying trials with certain recently-introduced fungicides. *J. Pomol.*, **23**, 185–205.

*MARSHALL, G. E. (1949). Interrelation of peach insects and disease control at Orleans, Indiana. *J. econ. Ent.*, **42**, 806–810.

MASON, C. L. (1949). The use of sodium hypochloride in the control of peach diseases. *Plant Dis. Reptr*, **33**, 319–320.

*MASSEE, G. (1893). *British Fungus Flora*, Vol. 3, p. 283. London.

*MASSEE, G. (1895). *British Fungus Flora*, Vol. 4, p. 281. London.

*MASSEE, G. (1902). Plant diseases. *J. R. hort. Soc.*, **26**, 736.

*MASSEE, G. (1903). *A Text-book of Plant Diseases*, pp. 300–302. London.

MASSEE, G. (1915). *Diseases of Cultivated Plants and Trees*, pp. 270–273. London.

*MASSEE, G. & CROSSLAND, C. (1905). *The Fungus Flora of Yorkshire*, pp. 278 and 328. London.

*MATHENY, W. A. (1913). A comparison of the American Brown-rot fungus with *Sclerotinia fructigena* and *S. cinerea* of Europe. *Bot. Gaz.*, **56**, 418–432.

MAYLIN, M. (1929). La destruction et la chute des fruits dues aux maladies cryptogamiques. *Vie agric. rur.*, **18**, 212–216.

*MCALPINE, D. (1902). *Fungus diseases of stone-fruit trees in Australia and their treatment*, pp. 53, 85. Melbourne.

*MCCALLAN, S. E. A. (1930). Studies on fungicides. II. Testing protective fungicides in the laboratory. III. The solvent action of spore secretions and other agencies on protective copper fungicides. *Mem. Cornell agric. Exp. Sta.*, 128, pp. 8–24; 25–79.

*MCCALLAN, S. E. A. (1948). Characteristic curve for the action of copper sulphate on the germination of spores of *Sclerotinia fructicola* and *Alternaria oleracea*. *Contr. Boyce Thompson Inst.*, **15**, 77–90.

MCCALLAN, S. E. A. (1951). Testing techniques. *Contr. Boyce Thompson Inst.*, **16**, 299–302.

MCCALLAN, S. E. A., MILLER, L. P. & WEED, R. M. (1952). Fungitoxicity of compounds possibly concerned in the mode of action of sulfur and ferbam. (Abstr.) *Phytopathology*, **42**, 14.

MCCALLAN, S. E. A. & WEEDON, F. R. (1940). Toxicity of ammonia, chlorine, hydrogen cyanide, hydrogen sulphide, and sulphur dioxide gases. II. Fungi and bacteria. *Contr. Boyce Thompson Inst.*, **11**, 331–342.

McCallan, S. E. A., Wellman, R. H. & Wilcoxon, F. (1941). An analysis of factors causing variation in spore germination tests of fungicides. III. Slope of toxicity curves, replicate tests, and fungi. *Contr. Boyce Thompson Inst.*, **12**, 49–77.

McCallan, S. E. A. & Wellman, R. H. (1942). Fungicidal versus fungistatic. *Contr. Boyce Thompson Inst.*, **12**, 451–463.

McCallan, S. E. A. & Wellman, R. H. (1943). Cumulative error terms for comparing fungicides by established laboratory and greenhouse methods. *Contr. Boyce Thompson Inst.*, **13**, 135–141.

*McCallan, S. E. A. & Wilcoxon, F. (1931). The fungicidal action of sulphur. II. The production of hydrogen sulphide by sulphured leaves and spores and its toxicity to spores. *Contr. Boyce Thompson Inst.*, **3**, 13–38.

McCallan, S. E. A. & Wilcoxon, F. (1932). The precision of spore germination tests. *Contr. Boyce Thompson Inst.*, **4**, 233–243.

*McCallan, S. E. A. & Wilcoxon, F. (1933). The form of the toxicity surface for copper sulphate and for sulphur, in relation to conidia of *Sclerotinia americana*. *Contr. Boyce Thompson Inst.*, **5**, 173–180.

McCallan, S. E. A. & Wilcoxon, F. (1934). Fungicidal action and the periodic system of the elements. *Contr. Boyce Thompson Inst.*, **6**, 479–500.

McCallan, S. E. A. & Wilcoxon, F. (1936). The action of fungous spores on Bordeaux mixture. *Contr. Boyce Thompson Inst.*, **8**, 151–165.

*McCallan, S. E. A. & Wilcoxon, F. (1938). Laboratory comparison of copper fungicides. *Contr. Boyce Thompson Inst.*, **9**, 249–263.

McCallan, S. E. A. & Wilcoxon, F. (1940*a*). An analysis of factors causing variation in spore germination tests of fungicides. I. Methods of obtaining spores. *Contr. Boyce Thompson Inst.*, **11**, 5–20.

*McCallan, S. E. A. & Wilcoxon, F. (1940*b*). An analysis of factors causing variation in spore germination tests of fungicides. II. Methods of spraying. *Contr. Boyce Thompson Inst.*, **11**, 309–324.

*McClellan, W. D. (1942). Temperature as it affects spore germination in the presence of copper and sulphur. *Phytopathology*, **32**, 394–398.

*McCubbin, W. A. (1918). Peach canker. *Bull. Dom. Can. Dep. Agric. Dom. exp. Fms.*, 37, 20 pp.

McCubbin, W. A. (1919). Brown rot of stone fruits. *Agric. Gaz. Can.*, **6**, 429–432.

*McNew, G. L. & Burchfield, H. P. (1950). Particle size in relation to the fungitoxicity of dichloronaphthoquinone. *Contr. Boyce Thompson Inst.*, **16**, 163–176.

*McNew, G. L. & Burchfield, H. P. (1951). Fungitoxicity and biological activity of quinones. *Contr. Boyce Thompson Inst.*, **16**, 357–374.

*McNew, G. L. & Sundholm, N. K. (1949). The fungicidal activity of substituted pyrazoles and related compounds. *Phytopathology*, **39**, 721–751.

McWhorter, O. T. (1941). Sulphur dust, spray controls brown rot. *Bett. Fruit*, **36**, pp. 5, 13.

*Meier, K. & Grampoloff, A. V. (1936). L'action des rayons ultra-violets sur l'entreposage des denrées périssables. *Annu. agric. Suisse*, **37**, 951–977.

*Menon, K. P. V. (1934). Studies in the physiology of parasitism. XIV. Comparison of enzymic extracts obtained from various parasitic fungi. *Ann. Bot., Lond.*, **48**, 187–210.

*Michener, H. D. & Snell, N. (1949). Two antifungal substances from *Bacillus subtilis* cultures. *Arch. Biochem., N.Y.*, **22**, 208–214.

Mieller, H. (1949). Über den Zuckergehalt von schwarzfaulen Äpfeln und Birnen. *Z. PflKrankh.*, **56**, 258–262.

*Mijuskhovits, M. (1950). [Diseases of plants recorded in Montenegro in 1949]. *Plant Protection, Belgrade*, 1950, No. 1, pp. 101–104.

Milbrath, D. G. (1935). Brown rot in deciduous fruit production. *Blue Anchor*, **12**, pp. 2–3, 19.

Miller, H. J. (1944). The use of *Venturia inaequalis* and *Sclerotinia fructicola* with pure chemical stimulants in slide-germination tests of fungicides. *Phytopathology*, **34**, 1009.

Miller, H. J. (1947). Factors influencing sporulation of *Sclerotinia fructicola*, *Venturia inaequalis* and *Phytophthora infestans*. (Abstr.) *Phytopathology*, **37**, 15–16.

Miller, H. J. (1949). Modifications of the slide-germination method of evaluating fungicides including the use of *Venturia inaequalis* and *Phytophthora infestans*. *Phytopathology*, **39**, 245–259.

*Miller, H. J. (1950). Relation of concentration of some organic substances to spore germination and dosage response. *Phytopathology*, **40**, 326–332.

Miller, P. R. (1949). Apple scab control by eradicant fungicides. *Agric. Chemicals*, **4**, pp. 47–49, 77–79.

Mills, W. D. (1945). Fruit diseases in 1944. *Proc. N.Y. St. hort. Soc. 90th ann. Meeting*, 21–30.

Mittmann, Gertrud. (1938). Infektionsversuche an Obstbäumen mit Stämmen verschiedener Herkunft von *Monilia cinerea* und *Monilia fructigena*. *Z. PflKrankh.*, **48**, 232–246.

Mittman-Maier, Gertrud. (1939). Die Monilia-Krankheiten der Obstbäume. *Geisenheimer Mitt.*, 7 pp.

*Mittmann-Maier, Gertrud. (1940*a*). Untersuchungen über die Monilia-resistenz von Sauerkirschen. *Z. PflKrankh.*, **50,** 84–95.

*Mittmann-Maier, Gertrud. (1940*b*). Untersuchungen über die Anfälligkeit von Äpfel- und Birnensorten gegenuber der Moniliafrucktfäule. *Gartenbauwiss.*, **15,** 334–361.

Mittmann-Maier, Gertrud. (1942). Monilia-Schaden an Obst. *Geisenheimer Mitt.*, **56,** 18–23.

*Miura, M. (1929). *Flora of Manchuria and East Mongolia. Part III. Cryptogams, Fungi*, pp. 93–98.

Mix, A. J. (1930*a*). A blight of flowering almond, *Prunus glandulosa* Thunb. *Phytopathology*, **20,** 265.

Mix, A. J. (1930*b*). Brown-rot leaf and twig blight following peach-leaf curl. *Phytopathology*, **20,** 265–266.

Miyushkovits, M. (1950). Diseases of plants noted in Montenegro in 1949. [Serb]. *Plant Protection, Belgrade*, No. 1, pp. 94–105.

*Molliard, M. Marie. (1901). Sur une epidémie de Rot Brun aux environs de Paris. *Bull. Soc. mycol. Fr.*, **17,** 280. (Abstr.) *J. R. hort. Soc.*, **26,** 1901–2, 908.

*Molz, E. (1907). Ueber die Bedingung der Entstehung der durch *Sclerotinia fructigena* erzeugten Schwarzfäule der Aepfel. *Zbl. Bakt.*, **17,** 175–188.

*Montemartini, L. (1924). Rassegna crittogamica per l'anno, 1921, 1922, 1923. *Atti Ist. bot. Univ. Pavia*, **1,** pp. IX–LXIII.

*Montemartini, L. (1936). [New observations on the parasites and diseases of plants grown in Western Sicily: three-year period, 1934–36]. *Riv. Pat. veg.*, **26,** 355–377.

*Moore, M. H. (1932). Further studies on the incidence and control of apple scab (*Venturia inaequalis*) and apple mildew (*Podosphaera leucotricha*) at East Malling. *J. Pomol.*, **10,** 271–294.

*Moore, M. H. (1933). Some incidental effects of routine scab-sprays, with special reference to apple fruit sawfly-control—a sidelight on the interpretation of field spraying experiments. *Rep. E. Malling Res. Sta. for* 1932, 90–98.

*Moore, M. H. (1947). Preliminary report on *Monilia fructigena* and *Botrytis cinerea* as wound parasites of cobnuts and filberts. *Rep. E. Malling Res. Sta. for* 1946, 120–121.

*Moore, M. H. (1950*a*). Nut rotting in *Corylus avellana* in relation to the activities of the nut weevil, *Balaninus nucum* L. *J. hort. Sci.*, **25,** 213–224.

*Moore, M. H. (1950*b*). Brown rot of apples: fungicide trials and studies of the relative importance of different wound-agents. *J. hort. Sci.*, **25,** 225–234.

*Moore, M. H. (1950*c*). Scab and codling sprays reduce brown rot loss. *Grower*, **33,** 368–371.

Moore, M. H. (1951). Brown rot of apples: note on infection associated with hail bruises and lenticels. *Rep. E. Malling Res. Sta. for* 1950, p. 131.

*Moore, M. H. (1952). Notes on infection of young peach shoots and of leaves by the brown rot fungus, *Monilia fructigena*. *Rep. E. Malling Res. Sta. for* 1951, p. 148.

*Moore, M. H. & Rogers, W. S. (1942). Sun scald of fruits. *Gdnrs' Chron.*, **112,** 166–167.

*Moore, M. H., Steer, W. & Shaw, H. (1939). Recent work on fungicides and insecticides at East Malling. *Sci. Hort.*, **7,** 85–95, and *Rep. E. Malling Res. Sta. for* 1938, pp. 253–261.

*Moore, W. C. (1951). Plant Diseases in the United Kingdom. *Proc. U.N. Scientif. Conf. Conservation and Utilization of Resources*, **6,** 325–7.

*Morgan, O. D. & Powell, D. (1949). The effect of wetting agents on brown rot control. (Abstr.) *Phytopathology*, **39,** 16.

Morse, W. J. & Lewis, C. E. (1910). Maine apple diseases. *Bull. Me agric. Exp. Sta.*, 185, p. 364.

*Morwood, R. B. (1931). The work of the Pathological Branch. *Rep. Qd Dep. Agric.*, 1930–1931, pp. 47–48.

*Muhleman, G. W. (1925). The pectinase of *Sclerotinia cinerea*. *Bot. Gaz.*, **80,** 325–330.

*Müller, A. S. (1941). [Survey of diseases of cultivated plants in Venezuela, 1937–1941]. *Bol. Soc. venez. Cien. nat.*, **7,** pp. 99–113. [*R.A.M.*, **21,** 324].

*Muller, A. S. (1950). A preliminary survey of plant diseases in Guatemala. *Plant Dis. Reptr*, **34,** 161–164.

*Müller-Thurgau, H. (1900). Die Monilienkrankheit oder Zweigdurre der Kernobstbaume. *Schweiz. Z. Obst- u. Weinb.*, Nos. 13, 14, pp. 198–203.

*Müller-Thurgau, H. & Osterwalder, A. (1924). Bericht der Schweizerischen Versuchsanstalt für Obst-, Wein- und Gartenbau in Wädenswil für die Jahre, 1921–1923. III. 1. Pflanzenphysiologische und pflanzenpathologische Abteilung. *Landw. Jb. Schweiz*, **38,** 573–581.

*Murphy, P. A. (1926). Some fungus disease of fruit trees. *J. Dep. Lands Agric., Dublin*, **25,** 269–280.

*Nagorny, P. I. & Issarlischwili, S. J. (1929). [Representatives of the fungal flora of the vine hitherto unknown in the Caucasus]. *Bull. Inst. Exp. Agric. Georgia, Tiflis*, **1,** 5–18. [*R.A.M.*, **9,** 429].

*Najjar, H. (1947). [The brown rot of apricots]. *Circ. Ext. Serv. Syrian Minist. nat. Economy, Damascus*, 3, 14 pp.

*Nannfeldt, J. A. (1932). Studien über die Morphologie und Systematik der nicht-lichenisierten inoperculaten Discomyceten. *Nova Acta Soc. Sci. upsal.*, 8, 368 pp.

*Noble, R. J., Hynes, H. J., McCleery, F. C. & Birmingham, W. A. (1934). Plant diseases recorded in New South Wales. *Sci. Bull. Dep. Agric. N.S.W.*, 46, 47 pp.

*Noble, R. J., Hynes, H. J., Magee, C. P., McCleery, F. C., Birmingham, W. A., Edwards, E. T. & Brown, H. P. (1937). The occurrence of plant diseases in New South Wales, with particular reference to the three-year period ending 30th June, 1936. *Sci. Bull. Dep. Agric. N.S.W.*, 57, p. 34.

*Norton, J. B. S. (1902). *Sclerotinia fructigena. Trans. Acad. Sci. St Louis*, **12**, 91–97.

*Norton, J. B. S. (1914). Hosts of brown-rot Sclerotinia. (Abstr.) *Phytopathology*, **4**, 398.

*Norton, J. B. S. & Ezekiel, W. N. (1924). The name of the American brown-rot *Sclerotinia*. (Abstr.) *Phytopathology*, **14**, 31–32.

*Norton, J. B. S., Ezekiel, W. N. & Jehle, R. A. (1923). Fruit-rotting Sclerotinias I. Apothecia of the brown-rot fungus. *Bull. Md agric. Exp. Sta.*, 271, 87–142.

*de Ong, E. R. (1932). Fungicidal value of pine-tar oil and copper resinate. *Phytopathology*, **22**, 861–864.

*O'Kane, W. C. (1950). Fungicidal composition—U.S. Patent 2460, 188. Application, May 2nd, 1949. (Abstr.) *Biol. Abstr.*, **24**, 1049.

Oppenheimer, H. R. (1926). Die Therapie der Baumschulkrankheit. *Angew. Bot.*, 8, 137–146.

*Osterwalder, A. & Kessler, H. (1934). Das Auftreten der Fäulnis und nicht parasitärer Krankheiten bei der Kühllagerung des Obstes. *Schweiz. Obst- u. Weinb. Wädenswil*, pp. 423–424.

*Ovtshinnikova, A. A. (1937). [Summary of the scientific research work of the Institute of Plant Protection for the year 1936. Part II. Pests and diseases of industrial and fruit crops]. *Publ. Off. Pan-Sov., V. I. Lenin Acad. agric. Sci., Leningrad*, pp. 376–378. [*R.A.M.*, **17**, 441].

*Palmiter, D. H. & Hamilton, J. M. (1942). A new fungicide. *Proc. N.Y. St. agric. Soc.*, 1942, 207–209.

*Palmiter, D. H. & Hamilton, J. M. (1943). Organic materials in pre-harvest sprays for cherries. *Phytopathology*, **33**, 683–690.

*Palmiter, D. H. & Hamilton, J. M. (1945). Results of field and greenhouse experiments with new fungicides on orchard fruits in 1944. *Proc. N.Y. St. hort. Soc.*, 1945, pp. 16–20.

*Palmiter, D. H. & Keitt, G. W. (1937). The toxicity of copper-lime-arsenic mixtures to certain phytopathogenic fungi grown on malt agar plates. *J. agric. Res.*, **55**, 439–451.

Parodi, E. (1936). Sulle cause della decadenza della cultura del Cacao all'Ecuador e possible remedi. *Agron. colon.*, **30**, 121–127.

*Peck, C. H. (1881). Report of the Botanist. *Rep. N.Y. State Mus. nat. Hist.*, **34**, 34–36.

*Peglion, V. (1921). Manifestazioni epidemiche di *Sclerotinia cinerea* nella regione Emiliana. *R. C. Accad. Bologna*, 1920–21, 6 pp.

*Perishits, M. M. (1950). [Contribution to a knowledge of parasitic fungi of plants cultivated in Slovenia, 1948]. *Plant Protection, Belgrade*, 1950, No. 2, p. 71.

*Persoon, C. H. (1796). *Observationes Mycologicae*, p. 26. Lipsiae.

*Persoon, C. H. (1801). *Synopsis Methodica Fungorum*, p. 63. Gottingae.

*Pesante, A. (1935). Existence de formes ou de races biologiques dans 'Stromatinia fructigena' et 'Stromatinia cinerea'. *Bol. Sez. ital. Soc. int. Microbiol.*, **7**, 383–388.

*Pesante, A. (1938). Prime ricerche sulla sistematica e sulla biologia della 'Stromatineae' parasite dei fruttiferi in Italia. *Ann. Fac. agrar. for. Firenze*, 1937–8, **1**, 395–424.

*Petersen, D. & Cation, D. (1949). Spray injury to sour cherry petals during warm weather. *Plant Dis. Reptr*, **33**, 479–480.

*Petersen, D. & Cation, D. (1950). Exploratory experiments on the use of acti-dione for the control of peach brown rot and cherry leaf spot. *Plant Dis. Reptr*, **34**, 5–6.

*Petersen, D. H. & Cation, D. (1951). Peach blossom blight control experiments. *Quart. Bull. Mich. agric. Exp. Sta.*, **33**, 218–222.

*Peterson, P. D. (1941). The spore-germination method of evaluating fungicides. *Phytopathology*, **31**, 1108–1116.

*Philp, G. L. (1930). Cherry culture in California. *Circ. Calif. agric. Extens. Serv.*, 46, 42 pp.

*Pohl, M. (1939). Die Schädigungen in Pflaumenanbau durch die Monilia. *Obst- u Gemüseb.*, **85**, 95–97.

*Pollock, J. B. (1909). Notes on plant pathology. *Rep. Mich. Acad. Sci.*, **11**, 48–54.

*Pollock, J. B. (1918). The longveity in the soil of the *Sclerotinia* causing the brown rot of stone fruit. *Rep. Mich. Acad. Sci.*, **20**, 279–280.

*Pont, W. (1950). Brown rot control experiments in the Stanthorpe District, 1948–49 and 1949–50. *Qd agric. J.*, **71**, 265–269.

*Porter, C. L. (1932). Mixed cultures of bacteria and fungi. *Proc. Ind. Acad. Sci.*, **41**, 149–152.

*Porter, J. M. (1935). The effect of phosphorescent and fluorescent minerals upon the growth of fungi in cultures. *Proc. Ind. Acad. Sci.*, **44**, 76–78.

Poulos, P. L. (1949). The use of sodium hypochlorite for the control of the brown rot disease of peach in Delaware. *Plant Dis. Reptr*, **33**, 413–415.

*Poulos, P. L. (1950). Peach brown rot: relative effectiveness of various spray applications for control in the orchard. (Abstr.) *Phytopathology*, **40**, 22–23.

*Poulos, P. L. & Heuberger, J. W. (1949). Peach brown rot: pre-harvest sprays and control in the orchard and in the package. (Abstr.) *Phytopathology*, **39**, 18.

*Powell, D. (1946). Copper 8-quinolinolate, a promising fungicide. *Phytopathology*, **36**, 572–573.

*Powell, D. (1951). Phygon XL for the control of peach blossom blight. *Plant Dis. Reptr*, **35**, 76–77.

*Powers, W. L. & Bollen, W. B. (1947). Control of cracking of fruit by rain. *Science*, **105**, 334–335.

Pratt, H. St. J. (1935). Fruit tree spraying in Queensland. *Fruit World, Melbourne*, **36**, 9.

Prew, H. A. (1952). Brown rot of stone fruit. *N.Z. J. Agric.*, **84**, 28.

*Quaintance, A. L. (1900). The brown rot of peaches, plums, and other fruits. (*Monilia fructigena*). *Bull. Ga agric. Exp. Sta.*, 50, 237–269.

*Reade, J. M. (1908). Preliminary notes on some species of Sclerotinia. *Ann. mycol., Berlin*, **6**, 114–115.

Reinking, O. A. (1950). Plant disease investigations at the New York State Agricultural Experiment Station, Cornell University, Geneva, New York. *Suppl. Plant Dis. Reptr*, 191, 104–111.

*Reuter, E. (1898). In Norwegen im Jahre 1896 aufgetretene Krankheits-Erscheinungen. *Z. PflKrankh*, **8**, 209–214.

Rice, W. H. (1929). Control of brown-rot in stone-fruits. The Henderson experiments with peach-trees, 1925–29. *N.Z. J. Agric.*, **39**, 97–100.

[Rice, W. H. & Makgill, R. H.] (1927). Control of brown-rot in stone fruits. Experiments with peach trees at Henderson, 1926–27 season. *N.Z.J. Agric.*, **35**, 236–238.

[Rice, W. H. & Makgill, R. H.] (1928). Control of brown-rot in stone fruits. Experiments with peach trees at Henderson, 1927–28 season. *N.Z. J. Agric.*, **36**, 419.

Rich, S. & Horsfall, J. G. (1950). Gaseous toxicants from organic sulfur compounds. *Amer. J. Bot.*, **37**, 643–650.

*Ritzema Bos, J. (1896). Ziekte in de Morellenboomen, veroorzaakt door *Monilia fructigena*. *Tijdschr. PlZiekt.*, **2**, 126–133.

*Ritzema Bos, J. (1898). Ziekte der vruchten en twijgen van den perzikboom, veroorzaakt door *Monilia fructigena* Persoon. *Tijdschr. PlZiekt*, **4**, 146–154.

*Ritzema Bos, J. (1904). Monilia-ziekten bij onze ooftboomen. *Tijdschr. PlZiekt.*, **9**, 125–145.

*Roberts, J. W. (1946). Recent developments in fungicides. II. Spray materials—1936–1944. *Bot. Rev.*, **12**, 538–547.

*Roberts, J. W. & Dunegan, J. C. (1924). The fungus causing the common Brown Rot of fruits in America. *J. agric. Res.*, **28**, 955–960.

*Roberts, J. W. & Dunegan, J. C. (1926). Blossom blight of the peach. *Phytopathology*, **16**, 217–222.

Roberts, J. W. & Dunegan, J. C. (1927*a*). Peach brown rot and scab. *Bull. U.S. Dep. Agric.*, 1527, 14 pp.

*Roberts, J. W. & Dunegan, J. C. (1927*b*). Critical remarks on certain species of *Sclerotinia* and *Monilia* associated with diseases of fruits. *Mycologia*, **19**, 195–205.

*Roberts, J. W. & Dunegan, J. C. (1932). Peach brown rot. *Tech. Bull. U.S. Dep. Agric.*, 328, 59 pp.

*Roberts, J. W. & Pierce, L. (1932). Zinc-lime: a fungicide for the peach. *Phytopathology*, **22**, 415–427.

Rodda, T. E. (1920). Control of brown-rot of peaches: the past season's experiments at Arataki. *N.Z. J. Agric.*, **21**, 20.

Rose, D. H. (1924*a*). Diseases of apples on the market. *Dep. Bull. U.S. Dep. Agric.*, 1253, 24 pp.

Rose, D. H. (1924*b*). Diseases of stone fruits on the market. *Frms' Bull. U.S. Dep. Agric.* 1435. 16 pp.

*Rose, D. H., Brooks, C., Fisher, D. F. & Bratley, C. O. (1933). Market diseases of fruits and vegetables: apples, pears and quinces. *Misc. Publ. U.S. Dep. Agric.* 168. pp. 14–15.

*Rose, D. H., Fisher, D. F., Brooks, C. & Bratley, C. O. (1937). Market diseases of fruits and vegetables: peaches, plums, cherries and other stone fruits. *Misc. Publ. U.S. Dep. Agric.* 228, pp. 5–8.

*Rose, D. H., Fisher, D. F., Brooks, C. & Bratley, C. O. (1950). Market diseases of fruits and vegetables: peaches, plums, cherries and other stone fruits. *Misc. Publ. U.S. Dep. Agric.* 228, 27 pp.

*Rosella, E. (1939). La lutte contre les ennemis des arbres fruitiers. *Vie agric. rur.*, **29,** 377–381.

Rosella, E. (1946). La coulure des fleurs et le desséchement des rameaux d'arbres fruitiers à noyaux. *Prog. agric. vitic.*, **126,** 73–74.

Rudolph, B. A. (1922). Apricot brown rot control. *Univ. of Calif. Correction to Circ.* 238, 2 pp.

*Rudolph, B. A. (1925). Monilia blossom blight (brown rot) of apricots. *Bull. Calif. agric. Exp. Sta.* 383, 55 pp.

Rudolph, B. A. (1935). Brown rot of stone fruits and its control under Californian conditions. *Blue Anchor*, **12,** 23–24.

*Rudolph, B. A. (1936). Brown rot of stone fruits on the Pacific coast and its control. *Bett. Fruit*, **30,** 3–5.

*Salmon, E. S. (1907). "Brown Rot" of acid cherries. *J. S.-E. agric. Coll. Wye*, **16,** 283–286.

*Salmon, E. S. (1910*a*). A "Canker" of apple trees caused by the "Brown Rot" fungus. *J. S.-E. agric. Coll. Wye*, **19,** 355–357.

*Salmon, E. S. (1910*b*). A "canker" of apple trees caused by the "Brown Rot" fungus. *Gndrs' Chron.*, **47,** 327.

*Salmon, E. S. (1913). The "Brown Rot" canker of the apple. *J. S.-E. agric. Coll. Wye*, **22,** 446–449. (Also in *Gdnrs' Chron.* **56,** 1914, p. 85.)

*Salmon, E. S. & Ware, W. M. (1931). Report from the Mycological Department. *J. S.-E. agric. Coll. Wye*, **28,** 48–56.

Salmon, E. S. & Ware, W. M. (1933). Department of Mycology. *J. S.-E. agric. Coll. Wye*, **31,** 13–21.

*Sardiña, J. R. (1928). Los preparados coloidales en terapéutica vegetal. *Bol. Pat. veg. Ent. agric. Madr.*, **3,** 24–30.

*Sarejanni, J. A. (1935). Sur le Sclerotinia de l'Amandier. *Ann. Inst. phytopath. Benaki*, **1,** 57–60. [*R.A.M.*, **15,** 591].

*Sarejanni, J. A. (1936). Notes phytopathologiques. II. *Ann. Inst. phytopath. Benaki*, **2,** 86–92. [*R.A.M.*, **18,** 10].

*Sarejanni, J. A. (1939). Catalogue commenté des champignons rencontrés sur les plantes cultivées en Grèce. *Ann. Inst. phytopath. Benaki*, **3,** 41–66. [*R.A.M.*, **28,** 51].

*Sattar, A. (1940). Some important diseases of apples in the Punjab and their control. *Punjab Fruit J.*, **4,** 719–721.

*Sauchelli, V. (1933). Flotation sulfur in agriculture. *Industr. Engng Chem.*, **25,** 363–367.

*Savulescu, T., Sandu-Ville, C., Hulea, A. & Hulpoi, A. (1941). Starea fitosanitară in România in anul 1938–1939. *Institutul de cercetări agronomice al României*, Nr. 72, Bukarest 106 pp. [*Z. PflKrankh.*, **54,** 319–320].

*Schellenberg, H. C. (1923). Infectionsversuche mit Vertretern der Gattung *Sclerotinia*. *Act. Soc. helv. Sci. nat.* 104, 161–162.

*Schenk, P. J. (1951). Monilia-ziekten van vruchtbomen. *Cult. Hand.*, **17,** 98–100.

*Schilberszky, K. (1933). Über die Ursachen der Apoplexie bei den Steinobstbaümen. II. *Angew. Bot.*, **15,** 106–122.

Schlösser, J. (1931). Schnitt der Schattenmorellen als Mittel zur Bekämpfung der Monilia. *Obst- u. Gemüseb.*, **77,** 77–78.

Schmidt, E. W. (1923). Ueber die fungizide Wirkung von Teerfarbstoffen. *Zbl. Bakt.*, **60,** 329–338.

Schmidt, E. W. (1924*a*). Wie kommt die Wirkung der Kupferkalkbrühe zustande? *Zbl. Bakt.*, **61,** 356–367.

*Schmidt, E. W. (1924*b*). Die fungizide Wirkung von Seifenlösungen. *Ber. dtsch. bot. Ges.*, **42,** 131–135.

*Schmidt, M. (1937). Infektionsversuche mit *Sclerotinia cinerea* an Süss- und Sauerkirschen. *Gartenbauwiss.*, **11,** 167–182.

*Schmidt, T. (1949). Das Auftreten wichtiger Krankheiten und Schädlinge an Kulturpflanzen in Österreich in Jahre 1946. *PflSch. Ber.* **3,** 48–54.

*Schmitt, C. G. (1951). Comparison of a series of derivatives of 4,5-dimethyl-2-mercaptothiazole for fungicidal efficacy. *Contr. Boyce Thompson Inst.*, **16,** 261–265.

Schneiderhan, F. J. & Hurt, R. H. (1925). The dry-mix spray for peaches. *Bull. Va agric. Exp. Sta.* 239, 16 pp.

*Schneider-Orelli, O. (1912). Versuche über die Wachstumsbedingungen und Verbreitung der Fäulnispilze des Lagerobstes. *Zbl. Bakt.*, **32,** 161–169.

*Schröter, C. (1893). *Krytogamen-Flora von Schlesien.* Bd. III, p. 67.

*Schubert, K. & Richter, K. (1926). Studien zur Bekämpfung des Apfelmehltaues (*Podosphaera leucotricha*) und einiger anderer Obstbaumschädlinge pilzliche und tierisher Art. *Angew. Bot.*, **8,** 146–147.

51238

*Schwarz, Marie B. (1922). Das Zweigsterben der Ulmen, Trauerweiden und Pfirschbäume. *Thesis Univ. Utrecht*, 73 pp.

de Schweinitz, L. D. (1822). Synopsis fungorum Carolinae superioris secundum observationes. *Soc. Nat. Cur. Lips.* 1; 20–131.

*Scott, W. M. & Ayres, T. W. (1910). The control of peach brown rot and scab. *Bull. U.S. Bur. Pl. Ind.* 174, 31 pp.

*Scott, W. M. & Quaintance, A. L. (1911). Spraying peaches for the control of brown-rot scab, and curculio. *Fmrs' Bull. U.S. Dep. Agric.* 440, 40 pp.

Scurti, F. & Pavarino, L. (1929). [On a new disease of peaches held in cold storage. The radiating disease.] *Ann. Chim. appl.*, **19**, 371–382. [*R.A.M.*, **9**, 255].

Seal, J. L. (1924). Biological specialization in *Sclerotinia* sp. the organism causing brown rot in fruits. *Minn. Studies Plant Sci., Studies Biol. Sci.* 5, 281–283.

*Seck, P. H. (1935). Die Monilia der Birne und des Apfels. *Obst- u. Gemüseb.*, **81**, 6—8.

Seidel, K. (1933). Zum Kampfgegen Monilia an Sauerkirschen. *Obst- u. Gemüseb.*, **79**, 174.

*Sharvelle, E. G. & Burkholder, C. L. (1947). Peach brown rot. *Hoosier Hort.*, **29**, 115–119.

Sharvelle, E. G. & Burkholder, C. L. (1949). The effect of low temperature exposure on the development of peach brown rot in common storage. (Abstr.) *Phytopathology*, **39**, 22.

*Sharvelle, E. G. & Chen, Shan-Ming. (1943). Cultural variation in single ascospore isolates of *Sclerotinia fructicola* (Wint.) Rehm from Cherry Plum hybrids. (Abstr.) *Phytopathology*, **33**, 1118.

*Sharvelle, E. G. & Teller, M. N. (1950). Control of brown rot. *Phytopathology*, **40**, 778–780.

Sharvelle, E. G. & Vandermark, J. S. (1952). Volatile chemicals for the inhibition of fruit losses in storage and transit. (Abstr.) *Phytopathology*, **42**, 19.

Shear, C. L. (1918). Pathological problems in the distribution of perishable plant produce. *Mem. Brooklyn bot. Gard.* 1, 415–422.

*Shear, C. L., Stevens, N. E. & Bain, H. F. (1931). Fungus diseases of the cultivated cranberry. *Tech. Bull. U.S. Dep. Agric.* 258, 57 pp.

*Sherbakoff, C. D. & Andes, J. O. (1936). Peach diseases and their control in Tennessee. *Bull. Tenn. agric. Exp. Sta.* 157, 11 pp.

Shima, Y. (1934). The influence of pollination on the outbreak of the apple young-fruit rot. *J. Sapporo Soc. Agr. & For.*, **26**, 173–182.

*Shima, Y. (1936). Studies on the young fruit-rot of apple-tree. *J. Fac. Hokkaido Univ.*, **39**, 143–270.

Shima, Z. (1934). Relations between pollination and stigmatic infection of young apple fruits by *Sclerotinia mali* Takahashi. *J. hort. Ass. Japan*, **5**, 8–12.

Shurtleff, M. C. & Shutak, V. G. (1952). Control of peach brown rot with the sodium salt of dehydroacetic acid. (Abstr.) *Phytopathology*, 42, 19.

*Shutak, V. & Christopher, E. P. (1948). Prolonging shelf life of plums with carbon dioxide. *Proc. Amer. Soc. hort. Sci.*, **51**, 231–234.

*Siemaszko, Wincenty. (1924). Pleśń lisciowa, *Monilia foliicola* Woronichin w swietle apostrzezeń i badań biologicznych. *Acta Soc. Bot. Polon.*, **2**, 81–98. [*Bot. Abstr.*, **15**, 781.]

*Siaens, F. (1951). Bespruitingen om het barsten van kersen te voorkomen. *Cult. Hand.*, **17**, 393.

*Simmonds, J. H. (1938). *Plant diseases and their control*, pp. 52–53. Dep. Agric. Qd Brisbane.

*Škorič, V. (1930). [Plant diseases at the University Farm "Maksimir".] *Rapp. Ann. Univ. de Zagreb* 1929, pp. 738–746. [*R.A.M.*, **9**, 625].

*Smarods, J. (1930). Larvijā novērotas kulturaugiem kaitigās sēnes. *Lauksaimn. Mēnešr.*, **10**, 31 pp.

*Smith, C. F. & Clayton, C. N. (1950). Peach spray information. *Special Circ. N.C. agric. Exp. Sta.* 5, 11 pp.

Smith, C. F. & Clayton, C. N. (1951). Peach spray information. *Special Circ. N.C. agric. Exp. Sta.* 12, 11 pp.

*Smith, E. F. (1889). Peach rot and peach blight. (*Monilia fructigena*, Persoon). *J. Mycol.* **5**, 123–134.

Smith, E. F. (1894). Peach blight, *Monilia fructigena*, Persoon. *J. Mycol.* 7, 36–38.

Smith, M. A. (1930*a*). Sulphur dust for the control of brown rot of peaches in storage. (Abstr.) *Phytopathology*, **20**, 122–123.

Smith, M. A. (1930*b*). The control of certain fruit diseases with flotation sulphurs. *Phytopathology*, **20**, 535–553.

*Smith, M. A. (1936*a*). Infection studies with *Sclerotinia fructicola.* (Abstr.) *Phytopathology*, **26**, 107.

*Smith, M. A. (1936*b*). Infection studies with *Sclerotinia fructicola* on brushed and non-brushed peaches. *Phytopathology*, **26**, 1056–1060.

*Smith, R. E. (1931). The life history of *Sclerotinia sclerotiorum* with reference to the green rot of apricots. *Phytopathology*, **21**, 407–423.

*Smith, W. G. (1885). Disease and decay in fruit. I. *Oidium fructigenum*. *Gdnrs' Chron.*, **24**, 51.

Smock, R. M. & Watson, R. D. (1941). Ozone in apple storage. *Refrig. Engng*, **42**, 97–101. [*R.A.M.*, **21**, 209].

*Smolák, J. (1929). Botrytis a Monilia na Révă Vinné. *Ochr. Rost.*, 9, 25–29. [*R.A.M.*, **9**, 13–14].

Snapp, O. I., Alder, C. H., Roberts, J. W., Dunegan, J. C. & Pressley, J. H. (1927). Experiments on the control of the plum curculio, brown rot, and scab, attacking the peach in Georgia. *Bull. U.S. Dep. Agric.* 1482, 32 pp.

Snapp, O. I., Turner, W. F. & Roberts, J. W. (1922). Controlling the curculio, brown-rot, and scab in the peach belt of Georgia. *Circ. U.S. Dep. Agric.* 216, 20 pp.

*Solkina, A. (1931). [The ascogenous stage of *Sclerotinia fructigena* in the neighbourhood of Leningrad.] [Russian.] *Pl. Prot. Leningr.*, **8**, 309–310. [*R.A.M.*, **11**, 310].

"Somerset". (1926). Brown rot and wither tip of plums (*Monilia cinerea* forma *pruni*). *Gdnrs' Chron.*, **78**, 17.

Sorauer, [P]. (1891). *Monilia fructigena*. *Z. PflKrankh.* **1**, 183.

*Sorauer, P. (1897). Ueber die jetzige Monilia—Epidemie der Kirschbäume. *Prakt. Ratg. Obstb.*, **12**, 281–282.

*Sorauer, P. (1899). Zur Monilia-Krankheit. *Ber. dtsch. bot. Ges.*, **17**, 186–189.

Sorauer. P. (1899–1900). Erkrankungsfälle durch *Monilia*. *Z. PflKrankh.* 9, 225–235; **10**, 148–154, and 274–284.

Sorauer, P. (1912). Weswegen erkranken Schatten-morellen besonders leicht durch Monilia. *Z. PflKrankh.* **22**, 285–292.

Sörensen, J. (1931). Mehr Schnitt im Schattenmorellenbau. *Obst- u. Gemüseb.*, **77**, 2, p. 23.

*Soriano, S. (1928). [Mycological notes on the cultivation in artificial media of some fungi parasitic on plants.] *Rev. Fac. Agron. B. Aires*, **6**, 89–114. [*R.A.M.*, **7**, 796].

*Spinks, G. T. (1916). A Black Rot of apples. *Rep. agric. hort. Res. Sta. Bristol*, 1915, 94–96.

*Spinks, G. T. (1917). A Black Rot of apples. *Rep. agric. hort. Res. Sta. Bristol*, 1916, 24–26.

*Sprau, F. (1948). Lagerschäden an Kernobst. *Pflanzenschutz*, **1**, 11–14.

*Staehelin, M. (1926). Beiträge zur Biologie und Physiologie der Obstbaumsclerotinien. *Verh. schweiz. naturforsch. Ges.* II. Teil, p. 206.

Staehelin, M. (1946*a*). Essais d'infection des fleurs de plusieurs espèces fruitieres avec les spores de *Monilia cinerea* de diverses origines. *Publ. Sta. fédér. d'essais vitic. Lausanne*, 351, pp. 764–765.

*Staehelin, M. (1946*b*). Station fédérale d'essais viticoles et arboricoles et de chimie agricole á Lausanne et à Pully. Rapport d'activité 1945. *Annu. agric. Suisse*, **47**, pp. 741–842.

*Staehelin, M. (1948). Gommose du pêcher en rapport avec le developpement du *Monilia fructigena*. *Landw. Jb. Schweiz*, **62**, 588.

Staehelin, M. & Wurgler, W. (1948). Gommose du pêcher en rapport avec le developpement du *Monilia fructigena*. *Annu. agric. Suisse*, **49**, 558.

*Steenland, A. P. (1949). Brown rot following leaf curl in Oregon. *Plant Dis. Reptr*, **33**, 203–204.

*Stevens, F. L. & Hall, J. G. (1921). *Diseases of economic plants*, 507 pp. New York.

Stevens, N. E. (1933). Some significant estimates of losses from plant diseases in the United States. *Phytopathology*, **23**, 975–984.

*Stevenson, A. W. (1926). Lime-sulphur affects canned cherries. *Bett. Fruit*, **21**, 10.

*Stewart, W. D. & Standen, J. H. (1946). Polymeric organic polysulphides as fungicides and spray adjuvants. *Contr. Boyce Thompson Inst.*, **14**, 203–220.

*Stoddard, E. M. (1950). Fungicidal synergism between urea and sulphur. (Abstr.) *Phytopathology*, **40**, 27.

*Strelin, S. L. (1926). [Grey rot of apricot (*Monilia laxa* Ehrenb.)] [Russian]. *Mater. Mikol. Fitopat. Ross.*, **5**, 177–186.

Strohbinder, M. F. & Driabina (Mme) M. M. (1935). [An experiment on the use of the Conn-Cholodny method for the microbiological investigation of vegetables and fruit during storage.] *Microbiology, Moscow*, **4**, 379–382.

*Susa, T. (1934). On the apple flower rot *Sclerotinia malicora* Miura and other important apple diseases. *Aormori hort. Exp. Sta. Kuroishi*, Aomori-Kiev, Japan.

Sushitski, L. A. (1950). [The preparation of copper naphthenate.] *Sad i Ogorod*, 1950, No. 2, pp. 25–26.

*Takahashi, Y. (1911). [On the Sclerotinia diseases of rosaceous fruit trees in Japan.] *Mirjabe-Festschrift*, pp. 135–155. Resumé in English in *Mykol. Zbl.*, **3**, 246–248, 1913.

*Talbot, F. M. (1949). Apricot growing in Central Otago. *N.Z. J. Agric.*, **78**, p. 151.

Tapke, V. F. (1946). A rapid method of isolating single ascospores from apothecia. *Phytopathology*, **36**, 167–168.

Tehon, L. R. & Stout, G. L. (1930). Epidemic diseases of fruit trees in Illinois 1922–1928. *Bull. Ill. Dep. Registr. Educ. Div. nat. Hist. Survey*, 18, pp. 415–502.

*Teller, M. N. & Mader, E. O. (1947). Changes in pectin produced by isolates of *Sclerotinia* spp. (Abstr.) *Phytopathology*, **37**, 21.

*Terrier, C. (1950). La "tacheture" des fleurs d'Azalées provoquée par le champignon *Ovulinia azaleae* Weiss; la présence de cette maladie en Suisse. *Rev. hort. Suisse*, **23**, 79–84.

*Thanos, A. (1951). Effect of ultraviolet light on the germination of spores of *Monilinia fructicola*, *Cladosporium cucumerinum*, and *C. epiphylum*. (Abstr.) *Phytopathology*, **41**, 35.

Thanos, A. (1952). Effect of cycloheximide (actidione) and some environmental factors on the germination of spores of *Monilinia fructicola* and *Botrytis cinerea*. (Abstr.) *Phytopathology*, **42**, 21.

*Thind, K. S. & Keitt, G. W. (1949). Studies on variability of *Sclerotinia fructicola* (Wint.) Rehm. *Phytopathology*, **39**, 621–636.

*von Thümen, F. (1875). Der Grind oder Schimmeldes Obstes (*Oidium fructigenum*). *Öst. landw. Wbl.*, pp. 41–48.

*Tilford, P. E. (1936). The relation of temperature to the effect of hydrogen- and hydroxyl-ion concentration on *Sclerotinia fructicola* and *Fomes annosus*. Spore germination and growth. *Bull. Ohio agric. Exp. Sta.* 567, 25 pp.

Tisdale, W. H. & Flenner, A. L. (1942). Derivatives of dithiocarbamic acid as pesticides. *Industr. Engng Chem.*, **34**, 501–502. [*R.A.M.*, **21**, 383–4].

*Tomkins, R. G. (1930). The effect of acetaldehyde on the growth of moulds. *Rep. Food Invest. Bd, Lond. for* 1929, pp. 57–59.

Tisdale, W. H. & Flenner, A. L. (1951). Derivatives of dithiocarbamic acid as fungicides *Contr. Boyce Thompson Inst.*, **16**, 303–311.

Triquart, J. (1933). Bekämpfung der Monilia auf Sauerkirschen. *Obst- u. Gemüseb.*, **79**, 73–74.

*Truesdell, W. H. (1922). Plant pathology in Crimea. *Phytopathology*, **12**, 533–535.

*Turček, F. J. (1950). [Does the pied-woodpecker control or distribute moniliosis.] *Ocht. Rost.*, **23**, 159–165.

*Unamuno, L. M. (1929). [New data relating to the study of the parasitic and saprophytic fungi of the environs of Durango (Vizcaya).] *Mem. R. Soc. Espanola Hist. nat.*, **15**, 345–354. [*R.A.M.*, **8**, 603–604].

*U.S. Department of Agriculture. (1944). Service and regulatory announcements. List of intercepted plant pests, 1943. *S.R.A.*, *Pl. Quarant. Adm.*, *Wash.*, 35 pp.

*U.S. Department of Agriculture. (1946). Service and regulatory announcements. List of intercepted plant pests, 1945. *S.R.A.*, *Pl. Quarant. Adm.*, *Wash.*, 47 pp.

*U.S. Department of Agriculture. (1950). Service and regulatory announcements. List of intercepted plant pests, 1948. *S.R.A.*, *Pl. Quarant. Adm.*, *Wash.*, 58 pp.

*Valleau, W. D. (1915). Varietal resistance of plums to brown rot. *J. agric. Res.*, **5**, 365–396.

Vandermark, J. S. & Sharvelle, E. G. (1952). Prevention of postharvest decay of stone fruits by volatile chemicals. *Science*, **115**, 149–50.

*Varma, Sardar, S. R. (1945). Pollination of plums in Patiala State hill orchards. *Punjab Fruit J.*, **9**, 134–6.

*Vasudeva, R. S. (1930*a*). Studies in the physiology of parasitism. XI. An analysis of the factors underlying specialization of parasitism with special reference to the fungi *Botrytis allii* Munn, and *Monilia fructigena* Pers. *Ann. Bot., Lond.*, **44**, 469–493.

*Vasudeva, R. S. (1930*b*). Studies in the physiology of parasitism. XII. On the effect of one organism in reducing the parasitic activity of another. *Ann. Bot., Lond.*, **44**, 557–563.

Vaughn, J. R. & Hamner, C. L. (1949). The present status of cycloheximide (Acti-dione) as a fungicide. *Proc. Amer. Soc. hort. Sci.*, **54**, 435–437.

*Vaughn, J. R., Lockwood, J. L., Randwa, G. S. & Hamner, C. (1949). The action of actidione on plant tissue and upon certain fungi. *Quart. Bull. Mich. agric. Exp. Sta.*, **31**, 456–464.

*Veresciaghin, B. (1940). Bolile cryptogamice pe ramurile si tulpinile pomilor roditori. *Bul. agric. Basarabia*, 1940, **1**, 11–12.

*Vielwerth, V. (1938). [Report on the agencies injurious to cultivated plants in western and central Slovakia.] *Ochr. Rost.*, **14**, 8–16. [*R.A.M.*, **17**, 501].

Vielwerth, V. & Slink-Mezencevová, A. (1938). [Experimental infection of Apricots with Monilia.] *Ochr. Rost.*, **14**, 47–50.

Voges, E. (1912). Über Monilia-Erkrankungen der Obstbäume. *Z. PflKrankh.*, **22**, 86–105.

*Voronichin, N. N. (1914). [Material for a mycological Flora of the Sochi district.] *Rep. Sochi. hort. agric. Exp. Sta. for 1914*, 1–74. (Abstr.) *Plant Diseases, St. Petersburg*, 8, 39–43. [Russian.]

Voss, G. (1915). Monilia an Obstbäumen. *Flugbl. Königl. landw. Akad. Bonn- Poppelsdorf.*, 7, 4 pp.

*Wade, G. C. (1949). Apricot diseases. *Tasm. J. Agric.*, **20**, 22–29.

*Wade, G. C. (1951). Progress report of brown rot of apricot investigations. *Tasm. J. Agr.*, **22**, 258–266.

*Wahl, B. (1924). Bericht über die Tätigkeit der Bundesanstalt für Pflanzenschutz in Wien in den Jahren 1921 bis 1923. *Z. landw. VersWes. Öst*, 48 pp. [*R.A.M.*, 4, 145].

*Wahl, B. (1925). Bericht über die Tätigkeit der Bundesanstalt für Pflanzenschutz in Wien im Jahre 1924. *Z. landw. VersWes. Öst.* 1925, 28 pp. [*R.A.M.*, **5**, 210].

*Wahl, B. (1926). Bericht über die Tätigkeit der Bundesanstalt für Pflanzenschutz in Wien im Jahre 1925. Verlag. *Bundesanst. Pflschutz. Wien II*, 28 pp. [*R.A.M.*, **6**, 530–1].

*Wallace, E., Blodgett, F. M. & Hesler, L. R. (1911). Studies of the fungicidal value of lime-sulphur preparations. *Bull. N.Y. (Ithaca) agr. Exp. Sta.*, 290 pp.

*Ward, J. M. (1926). Prune and plum culture. Fungoid diseases. *J. Dep. Agric. Vict.*, **24**, 490–493.

*Warren, J. R., Graham, F. & Gale, G. (1951). Dominance of an actinomycete in a soil microflora after 2,4-D treatment of plants. *Phytopathology*, **41**, 1037–1039.

Watkins, –, Morris, H. F. & Langley –. (1942). Peach diseases. *Ann. Rep. Tex. agric. Exp. Sta.* 1941, p. 73.

*Watson, R. D. (1943). Some factors influencing the toxicity of ozone to fungi in cold storage. *Refrig. Engng*, **46**, 103–106. [*R.A.M.*, **23**, 67].

*Waterston, J. M. (1947). The fungi of Bermuda. *Bull. Dep. Agric. Bermuda*, 23, p. 39 and 132.

Weaver, L. O. (1943*a*). Effect of temperature and relative humidity on occurrence of blossom blight of stone fruits. (Abstr.) *Phytopathology*, **33**, 15.

Weaver, L. O. (1943*b*). Effect of temperature and relative humidity on occurrence of blossom blight of stone fruits. *Cornell Univ. Thesis* (Abstract in *Cornell Univ. Abs. Theses*, 1943, 351–353, 1944).

*Weaver, L. O. (1948). Observations on the use of lime sulfur sprays on peach in Maryland, 1948. *Plant. Dis. Reptr*, **32**, 515–517.

*Weaver, L. O. (1950). Effect of temperature and relative humidity on occurrence of blossom blight of stone fruits. *Phytopathology*, **40**, 1136–1153.

*Weber, Anna. (1926). Sprøjtning af Frugttraeer og Frugtbuske mod Synltesvampe samt disses Biologi. *Tidsskr. Planteavl*, **32**, 219–318.

*Weber, Anna. (1938). Aeblesygdomme i lagerrum. *Tidsskr. Planteavl*, **43**, 337–345.

Wehmer, C. (1896). Pilzkrankheiten land- und forstwirtschaftlichen Kulturgewächse im Hannoverschen während des Sommers 1896. *Zbl. Bakt.*, **2**, 780–797.

Wehmer, C. (1898). *Monilia fructigena* Pers. (= *Sclerotinia fructigena* M.) und die Monilia-Krankheit der Obstbäume. *Ber. dtsch. bot. Ges.*, **16**, 298–307.

Wehmer, C. (1899). Entgegung auf die "Berichtigung" von B. Frank, *Monilia fructigena* betreffend. *Ber. dtsch. bot. Ges.*, **17**, 74–76.

*Wei, C. T. (1941). Notes on the storage and market diseases of fruits and vegetables. *Sinensia*, **12**, 135–152.

*Weindling, R. (1941). Experimental consideration of the mold toxins of *Gliocladium* and *Trichoderma*. *Phytopathology*, **31**, 991–1003.

Weiss, J. (1918). Einfluss der Willerungsverhältnisse auf das Auftreten von Pflanzenkrankheiten und tierischen Schädlingen 1916 und 1917. *Z. PflKrankh.*, **28**, 116–142, 201–210.

*Wellman, R. H. & McCallan, S. E. A. (1942). An analysis of factors causing variation in spore germination tests of fungicides. IV. Time and temperature. *Contr. Boyce Thompson Inst.*, **12**, 431–449.

*Wellman, R. H. & McCallan, S. E. A. (1943). A system for classifying effectiveness of fungicides in exploratory tests. *Contr. Boyce Thompson Inst.*, **13**, 171–176.

*Wellman, R. H. & McCallan, S. E. A. (1946). Glyoxalidine derivatives as foliage fungicides. I. Laboratory studies. *Contr. Boyce Thompson Inst.*, **14**, 151–160.

Wenzl, H. (1948). Zweigschäden durch *Sclerotinia laxa* an Marille. *PflSch. Ber., Wien.*, **2**, 140–144.

Wenzl, H. (1950*a*.) Steigerung der fungiziden wirksamkeit von Winterspritzmittelen in Frostspritverfahren. *PflSch. Ber., Wien.*, **4**, 110–116.

*Wenzl, H. (1950*b*). Untersuchungen über die Absterbeerscheinungen an Marille (*Prunus armeniaca*). *PflSch. Ber., Wien*, **4**, 187–200.

*Wenzl, H. (1951). Versuche zur Winterbekämpfung der Moniliakrankheit. *PflSch. Ber., Wien*, **6**, 178–189.

*Wernham, C. C. & Miller, H. J. (1948). Longevity of fungus cultures under mineral oil. *Phytopathology*, **38**, 932–934.

Werth, E. (1921). Phänologie und Pflanzenschutz. *Z. PflKrankh.*, **31**, 81–89.

*WESTERDIJK, JOHA. (1912). Der Sclerotinia der Kirsche. *Meded. phytopath. Lab. Scholten*, 3, 39–41.

*WHALEY, F. R. & HARRY, J. B. (1951). Chromate complexes as fungicides. *Contr. Boyce Thompson Inst.*, **16**, 375–386.

WHETZEL, H. H. (1912). The fungous diseases of the peach. *Proc. N.Y. State Fruit Grws' Assoc.*, **11**, 211–219.

*WHETZEL, H. H. (1945). A synopsis of the genera and species of the Sclerotiniaceae, a family of stromatic inoperculate Discomycetes. *Mycologia*, **37**, 648–714.

*WIESMANN, R. (1932). Untersuchungen über die Ueberwinterung des Apfelschorfpilzes *Fusicladium dendriticum* (Wallr.) Fckl. im toten Blatt, sowie die Ausbreitung der Sommersporen (Konidien) des Apfelschorfpilzes. *Landw. Jb. Schweiz*, **46**, 620–679.

WILCOX, E. M. (1905). Diseases of apple, cherry, peach, pear and plum, with methods of treatment. *Bull. Ala agric. Exp. Sta.*, 132, 142 pp.

WILCOX, J. C. (1950). Sprinkler irrigation experience in British Columbian orchards. *Sci. Agric.*, **30**, 418–427.

*WILCOXON, F. & MCCALLAN, S. E. A. (1930). The fungicidal action of sulphur: I. The alleged rôle of pentathionic acid. *Phytopathology*, **20**, 391–417. and *Contr. Boyce Thompson Inst.*, **2**, 389–417.

*WILCOXON, F. & MCCALLAN, S. E. A. (1931). The fungicidal action of sulphur. III. Physical factors affecting the efficiency of dusts. *Contr. Boyce Thompson Inst.*, **3**, 509–528.

*WILCOXON, F. & MCCALLAN, S. E. A. (1932). The fungicidal action of sulphur. IV. Comparative toxicity of sulphur, selenium and tellurium. *Contr. Boyce Thompson Inst.*, **4**, 415–424.

*WILCOXON, F. & MCCALLAN, S. E. A. (1935). Fungicidal action of organic thiocyanates, resorcinol derivatives and other organic compounds. *Contr. Boyce Thompson Inst.*, **7**, 333–339.

WILCOXON, F. & MCCALLAN, S. E. A. (1939). Theoretical principles underlying laboratory toxicity tests of fungicides. *Contr. Boyce Thompson Inst.*, **10**, 329–338.

WILKINSON, E. H. (1939). A note on the prevalence of fungal spots and rots of apples in cold store at Long Ashton. *Rep. agric. hort. Res. Sta. Bristol*, 1938, 84–90.

*WILLAMAN, J. J. (1920*a*). The function of vitamine in the metabolism of *Sclerotinia cinerea*. *J. Amer. chem. Soc.*, **42**, 551–585.

*WILLAMAN, J. J. (1920*b*). Pectin relations of *Sclerotinia cinerea*. *Bot. Gaz.*, **70**, 221–229.

*WILLAMAN, J. J. (1926). Biochemistry of plant diseases. VII. Correlation between skin texture and flesh texture in plum varieties. *Proc. Soc. exp. Biol., N.Y.*, **23**, 680–681.

WILLAMAN, J. J. & DAVISON, F. R. (1922). Biochemistry of plant diseases. II. Proximate analysis of plums rotted by *Sclerotinia cinerea*. *Bot. Gaz.*, **74**, 104–109.

*WILLAMAN, J. J., PERVIER, N. C. & TRIEBOLD, H. O. (1925). Biochemistry of plant diseases. V. Relation between susceptibility to brown rot in plums and physical and chemical properties. *Bot. Gaz.* **80**, 121–144.

WILLAMAN, J. J. & SANDSTROM, W. M. (1922). Biochemistry of plant diseases. III. Effect of *Sclerotinia cinerea* on plums. *Bot. Gaz.*, **73**, 287–307.

WILLAUME, M. F. (1949). Les traitements anticryptogamiques du pêcher, de l'abricotier et des arbres à fruits à noyaux. *Vitic. Arboric., Paris*, **95**, 5–7.

*WILLISON, R. S. (1931). Peach canker investigations. *Rep. Dom. Botanist for* 1930, *Div. Bot. Canad. Dep. Agric.*, pp. 116–120.

*WILLISON, R. S. (1932). Wound-gum in peaches and grapes. Its relation to the invasion of fungus wound-parasites. *Sci. Agric.*, **12**, 402–419; 484–505.

*WILLISON, R. S. (1933). Peach canker investigations. I. Some notes on incidence, contributing factors, and control measures. *Sci. agric.*, **14**, 32–47.

*WILLISON, R. S. (1935). Inoculation studies in peach canker. (Abstr.) *Sci. Agric.*, **15**, 435.

*WILLISON, R. S. (1937). Controlling brown rot of peaches in transit. *Canad. Hort.*, **60**, 7.

*WILLISON, R. S. (1939). Brown rot of peaches in transit and storage. *Sci. Agric.*, **19**, 458–474.

*WILLISON, R. S. (1944). Brown rot and other fungal wastage in harvested peaches. *Sci. Agric.* **24**, 221–233.

*WILLISON, R. S. & CHAMBERLAIN, G. C. (1935). Studies in fruit diseases. VIII. Preventing peach canker. *Circ. Dep. Agric. Can.*, 92, 8 pp.

*WILSON, E. E. (1939). The effect of petroleum oil emulsions on the fungicidal value of Bordeaux mixture. (Abstr.) *Phytopathology*, **29**, 828.

*WILSON, E. E. (1940). Eradicant sprays for the control of blossom infection by *Sclerotinia laxa*. (Abstr.) *Phytopathology*, **30**, 27.

WILSON, E. E. (1942). Experiments with arsenite sprays to eradicate *Sclerotinia laxa* in stone fruit trees as a means of controlling the brown rot disease in blossoms. *J. agric. Res.*, **64**, 561–594.

WILSON, E. E. (1943*a*). Tests of eradicant sprays for use against *Sclerotinia laxa* and *Coryneum beijerinckii* in apricots and almonds. *Phytopathology*, **33**, 506–516.

Wilson, E. E. (1943*b*). The chlorophenates as eradicant sprays against *Sclerotinia laxa* and *Coryneum beijerinckii*. (Abstr.) *Phytopathology*, **33**, 1120.

Wilson, E. E. (1948). Relation of temperature and moisture to the effectiveness of sodium pentachlorophenate as an eradicant spray against *Sclerotinia laxa*. (Abstr.) *Phytopathology*, **38**, 919.

Wilson, E. E. (1949). Effect of bordeaux and petroleum oil on the eradicative action of monocalcium arsenite against *Sclerotinia laxa* in apricot trees. *Plant Dis. Reptr*, **33**, 287–289.

*Wilson, E. E. (1950). Sodium pentachlorophenate and other materials as eradicative fungicides against *Sclerotinia laxa*. *Phytopathology*, **40**, 567–583.

*Wilson, E. E. & Baker, G. A. (1946*a*). Some features of the spread of plant diseases by air-borne and insect-borne inoculum. *Phytopathology*, **36**, 418–432.

*Wilson, E. E. & Baker, G. A. (1946*b*). Some aspects of the aerial dissemination of spores, with special reference to conidia of *Sclerotinia laxa*. *J. agric. Res.*, **72**, 301–327.

*Wilson, E. E. & Serr, E. F. (1938). Preliminary tests to determine effect of arsenite sprays on sporodochia of *Sclerotinia laxa* and on control of brown rot in blossoms of almond and apricot. *Phytopathology*, **28**, 759–760.

*Wilson, E. E. & Scott, C. E. (1943*a*). Prevention of three peach diseases by ferric dimethyldithiocarbamate spray. *Phytopathology*, **33**, 962–963.

*Wilson, E. E. & Scott, C. E. (1943*b*). Dormant spraying with $Ca(AsO_2)_2$ for the control of brown rot blossom blight in apricot trees. *Blue Anchor*, **20**, 27.

*Wiltshire, S. P. (1920). The production of conidia in pure cultures by the brown rot fungus of the apple. *Rep. agric. hort. Res. Sta. Bristol*, 1919, 34–36.

*Winter, G. (1883). Ueber einige nordamerikanische Pilze. *Hedwigia*, **22**, 67–72, 129–131.

(Winter, H. F. & Young, H. C.) (1933). Fifty-first Annual Report of the Ohio Agricultural Experiment Station for the year ended June 30, 1932. *Bull. Ohio agric. Exp. Sta.* 516.

*Winter [H. F.] & Young, [H. C.]. (1938). Progress of agricultural research in Ohio 1936–1937. Botany and plant pathology. *Bull. Ohio agric. Exp. Sta.* 592, p. 35.

*Wood, M. N. (1937). Almond culture in California. *Circ. Calif. agric. Ext. Serv.* 103, 96 pp.

*Wormald, H. (1917*a*). A blossom wilt and canker of apple trees. *Ann. appl. Biol.*, **3**, 159–204.

*Wormald, H. (1917*b*). The "blossom wilt and canker" disease of apple trees. *J. Bd. Agric.*, **24**, 504–513.

*Wormald, H. (1918*a*). "Brown rot" of apples. *J. Bd. Agric.*, **25**, 299–302.

*Wormald, H. (1918*b*). A "wither tip" of plum trees. *Ann. appl. Biol.*, **5**, 28–59.

*Wormald, H. (1919). The "brown rot" diseases of fruit trees, with special reference to two biologic forms of *Monilia cinerea* Bon. I. *Ann. Bot., Lond.*, **33**, 361–404.

*Wormald, H. (1920*a*). The "brown rot" diseases of fruit trees, with special reference to two biologic forms of *Monilia cinerea* Bon. II. *Ann. Bot., Lond.*, **34**, 143–171.

*Wormald, H. (1920*b*). On the occurrence in Britain of the conidial stage of *Sclerotinia Mespili* Schell. *Ann. appl. Biol.*, 7, 173–177.

*Wormald, H. (1921). On the occurrence in Britain of the ascigerous stage of a "Brown Rot" fungus. *Ann. Bot., Lond.*, **35**, 125–135.

*Wormald, H. (1922*a*). Further studies of the Brown Rot fungi. I. A shoot-wilt and canker of plum trees caused by *Sclerotinia cinerea*. *Ann. Bot., Lond.*, **36**, 305–320.

*Wormald, H. (1922*b*). Observations on a discomycete found on medlar fruits. *Trans. Brit. mycol. Soc.*, **7**, 287–293.

*Wormald, H. (1923). Blossom wilt of plum trees. *J. Minist. Agric.*, **30**, 360–363.

*Wormald, H. (1926). On the occurrence in Britain of the conidial stage of *Sclerotinia cydoniae* Schell. *Trans. Brit. mycol. Soc.*, **10**, 303–306.

*Wormald, H. (1927*a*). A contribution to our knowledge of the distribution of the species of *Sclerotinia* causing brown rot. *Ann. Bot., Lond.*, **41**, 287–299.

*Wormald, H. (1927*b*). Brown rot of apples: the need for careful picking and sorting. *J. Minist. Agric.*, **34**, 552–554.

*Wormald, H. (1928*a*). Further studies of the brown-rot fungi. III. Nomenclature of the American brown-rot fungi: a review of literature and critical remarks. *Trans. Brit. mycol. Soc.*, **13**, 194–204.

*Wormald, H. (1928*b*). The present distribution of the brown rot fungi: its economic significance. *J. Minist. Agric.*, **35**, 741–750.

*Wormald, H. (1928*c*). Notes on Plant Diseases in 1926. *Rep. E. Malling Res. Sta. for* 1927, p. 86.

*Wormald, H. (1930*a*). Further studies of the brown rot fungi. IV. *Sclerotinia fructigena* as the cause of an apple canker. *Trans. Brit. mycol. Soc.*, **15**, 102–107.

*Wormald, H. (1930*b*). Further studies of the brown rot fungi. V. Brown-rot blossom wilt of pear trees. *Ann. Bot. Lond.*, **44**, 965–974.

*Wormald, H. (1931). Further studies of the brown rot fungi. VI. Brown rot blossom wilt of the Morello cherry: infection through unopened flowers. *J. Pomol.*, **9**, 232–237.

*Wormald, H. (1932). Blossom wilt of fruit trees. *J. Minist. Agric.*, **39**, 620–626.

*Wormald, H. (1933). Apples should be picked with their stalks. *J. Minist. Agric.*, **40**, 586–587.

Wormald, H. (1935). Further studies of the brown-rot fungi. VII. A shoot wilt in stools and layer beds of plum stocks, and its relation to wither tip. *J. Pomol.*, **13**, 68–77.

*Wormald, H. (1939). Notes on plant diseases in 1938. *Rep. E. Malling Res. Sta. for* 1938, 167–172.

*Wormald, H. (1940). Host plants of the brown rot fungi in Britain. *Trans. Brit. mycol. Soc.*, **24**, 20–28.

*Wormald, H. (1944). Nut drop: a disease of cultivated hazel nuts. *Gdnrs' Chron.*, **115**, 60–61, and *Rep. E. Malling Res. Sta. for* 1943, 56–58.

*Wormald, H. (1945). A black apple rot caused by *Monilia cinerea*. *Gdnrs' Chron.*, **117**, 115 and *Rep. E. Malling Res. Sta. for* 1944, 75–76.

*Wormald, H. & Moore, M. H. (1945). The control of brown rot of apples in commercial orchards. *Rep. E. Malling Res. Sta. for* 1944, 124–126.

*Wormald, H. & Painter, A. C. (1935). Brown rot of plums in cold storage. *Rep. E. Malling Res. Sta. for* 1934, 148–150.

*Wormald, H. & Painter, A. C. (1937). Further observations on brown rot of plums in cold storage. *Rep. E. Malling Res. Sta. for* 1936, 198–200.

*Woronin, M. (1888). Über die Sclerotienkrankheit der Vaccinieen-Beeren. Entwickelsgeschichte der diese Krankheit verursachenden Sclerotinien. *Mém. Acad. Sci. St.-Petersb. VIIe sér.*, 36, No. 6, 1–49.

*Woronin, M. (1895). Die Sclerotienkrankheit der gemeinen Traubenkirsche und der Eberesche (*Sclerotinia Padi* und *Sclerotinia Aucuparia*). *Mém. Acad. Sci. St.-Petersb. VIIIe sér.* 11, No. 1, Phys. Math., 1–27.

*Woronin, M. (1897). Kurze Notiz über *Monilia fructigena* Pers. *Z. PflKrankh*, **7**, 196–198.

*Woronin, M. (1900). Über *Sclerotinia cinerea* und *Sclerotinia fructigena*. *Mém. Acad. Sci. St.-Petersb. VIIIe sér.*, 10, Phys. Math., 1–38.

*Wulff, T. (1906). Stenfruktmögel. *Flygb. Kungl. Landsbruks-Akad.*, 5, 4 pp.

*Yarwood, C. E. (1936). The tolerance of *Erysiphe polygoni* and certain other powdery mildews to low humidity. *Phytopathology*, **26**, 845–859.

*Yarwood, C. E. (1948). Apricot jacket rot. (Abstr.) *Phytopathology*, **38**, 919–920.

Yarwood, C. E. (1950). Water content of fungus spores. *Amer. J. Bot.*, **37**, 636–639.

Yoder, D. M. (1951). Reversibility of copper toxicity to conidia of *Sclerotinia fructicola*. (Abstr.). *Phytopathology*, **41**, 39.

*Youganova, O. N. (1946). [Grey rot of apricot (Monilia) and measures for controlling it.] *Publication Crimean Res. Inst. Plant Prot.*, 71 pp.

*Youganova, O. N. (1951). [The effect of method of preparing and time of keeping copper naphthenate on its adherence when washed with water, its discoloration and toxicity.] *Doklady vsesojuz. Akad. seljsk. Nauk S.S.S.R.*, **16**, pp. 34–37.

*Youganova, O. N. & Sushitski, L. A. (1950). [The use of copper naphthenate in stone fruit disease control.] *Sad i Ogorod*, 1950, No. 12, pp. 30–33.

*Young, H. C. (1922). The toxic property of sulphur. *Ann. Mo. bot. Gdn.*, **9**, 403–433.

*Young, H. C. (1923). Sulphur as a fungicide. I. Colloidal sulfur. (Abstr.). *Phytopathology*, **13**, 60.

*Young, H. C. & Williams, R. (1928). Pentathionic acid: the fungicidal factor of sulphur. *Science*, **67**, 1920.

Zappe, M. P. & Stoddard, E. M. (1923). Results of dusting versus spraying in Connecticut apple and peach orchards in 1922. *Bull. Conn. agric. Exp. Sta.*, 245, 229–243.

Zaprometoff, N. G. (1925). [*Diseases of cultivated plants in middle Asia.*] 165 pp. Tashkent. [*R.A.M.*, **5**, 175].

*Zeller, S. M., Owens, C. E. & Evans, A. W. (1942). Experiments on the control of brown rot of cherries and peaches 1942, including notes on brown rot control in apricots. Progress report. *Ann. Rep. Ore. St. hort. Soc.*, 1942, pp. 53–57.

*Zimmermann, A. (1927). Sammelreferate über die Beziehungen zwischen Parasit und Wirtspflanze. *Zbl. Bakt.*, **69**, 352–425; **70**, 51–86; 261–313; 411–436.

APPENDIX

Many papers on various phases of the Brown Rot diseases have been noted while this bulletin was in the press; some of the more important ones are listed below.

Angell, H. R. (1952). Brown rot of stone fruits. I. An unsuccessful attempt to increase infection by artificial inoculation. *J. Aust. Inst. agric. Sci.*, **17**, 212–213.

Anon. (1951). Nuevas determinaciones micológicas para Chile. *Agric. tec., Chile*, **11**, 86–90. [*R.A.M.*, **31**, 354]

Brien, R. M. & Dingley, Joan M. (1951). A revised list of plant diseases recorded in New Zealand. *Bull. Dep. sci. industr. Res., N.Z.*, 101, 62 pp.

Brooks, R. M. & Olmo, H. P. (1951). Register of new fruit and nut varieties. *Proc. Amer. Soc. hort. Sci.*, **58**, 386–407. [*R.A.M.* **31**, 333]

Byrde, R. J. W. (1952). Experiments on the control of brown rot of apples and plums. II. Winter spraying trials. *J. hort. Sci.*, **27**, 192–200.

Byrde, R. J. W. (1952). Experiments on the control of brown rot of apples and plums. III. Summer spraying trials. *J. hort. Sci.*, **27**, 237–244.

Byrde, R. J. W., Crowdy, S. H. & Woodcock, D. (1953). Studies on systematic fungicides. III. The activity of certain chlorine-substituted β naphthols and naphthyloxy-*n*-aliphatic carboxylic acids as systemic fungicides. *Ann. appl. Biol.*, **40**, 152–165.

Gallucci, Maria M. (1952). Un infezione da *Monilia fructigena* Pers. su drupe di *Prunus laurocerasus* L. *Ann. Sper. agr., N.S.*, **6**, 1399–1404.

Hårdh, H. J. E. (1951). The pathogenicity of brown rot and its control by a parathion preparation. *Maataloust. Aikakausk.*, **23**, 79–87. [*R.A.M.* **32**, 83]

Kirby, R. S. (1952). Mercury sprays give indication of reducing bacterial leaf spot of peach foliage. *Plant Dis. Reptr*, **36**, 68.

Kittleson, A. R. (1952). A new class of organic fungicides. *Science*, **115**, 84–86.

Lovisolo, O. (1951). Attacchi di "*Monilia fructigena*" Pers. e di "*Monilia laxa*" Ehr. sui frutti di Nocciuolo. *Atti Accad. Torino*, **85**, (1950–1951). [*R.A.M.* **31**, 438]

Miller, L. P., McCallan, S. E. A. & Weed, R. M. (1953). Rate of uptake and toxic dose on a spore weight basis of various fungicides. *Contr. Boyce Thompson Inst.*, **17**, 173–195.

Miller, L. P., McCallan, S. E. A. & Weed, R. M. (1953). Quantitative studies on the role of hydrogen sulfide formation in the toxic action of sulfur to fungus spores. *Contr. Boyce Thompson Inst.*, **17**, 151–171.

Moore, M. H. & Talboys, P. W. (1953). Note on infection of blackberry fruits by the brown rot fungus *Monilia fructigena*. *Rep. E. Malling Res. Sta. for* 1952, 136–137.

Morgan, O. D. Jr. (1952). Some effects of eight fungicides on growth of *Sclerotinia fructicola*, *Glomerella cingulata* and *Penicillium expansum*. (Abstr.) *Phytopathology*, **42**, 147.

Owens, R. G. (1952). Spore uptake of fungitoxicants assayed by amylase inhibition. (Abstr.) *Phytopathology*, **42**, 471–472.

Owens, R. G. (1952). Chemical constitution of quinones in relation to amylase inhibition and fungitoxicity. (Abstr.) *Phytopathology*, **42.** 471.

Poulos, P. L. & Heuberger, J. W. (1952). Relation of wounds to the fruit rot phases of the brown rot disease of peaches. *Plant Dis. Reptr*, **36**, 198–200.

Roder, W. E., Monroe, C. M. & Whetstone, R. R. (1952). Tetrahydropyrimidine derivatives as potential foliage fungicides. *Science*, **115**, 124–125.

Ramsfjell, T. & Gjaesrum, H. B. (1950). [Experiments in the control of fruit and small fruit diseases in 1949.] *Meld. plPat. Inst., Oslo*, **4**, 32 pp.

Ribaldi, M. (1950). Influenza della temperatura sullo sviluppo della *Monilia fructigena* Pers. su frutte di Mela. *Riv. Ecol.*, **1**, 11 pp. [*R.A.M.* **31**, 437]

Sharvelle, E. G. & Burkholder, C. L. (1951). Evaluation of fungicides for the prevention of peach brown rot. *Bull. Ind. agric. Exp. Sta.* 564, 19 pp. [*R.A.M.* **31**, 389]

Sharvelle, E. G. & Vandemark, J. S. (1952). Volatile chemicals for the inhibition of fruit losses in storage and transit. (Abstr.) *Phytopathology*, **42**, 19.

Standen, J. H. (1952). Host index of plant pathogens of Venezuela. *Suppl. Plant Dis. Reptr.*, 212, 106 pp.

Vandemark, J. S. & Sharvelle, E. G. (1952). Prevention of post-harvest decay of stone fruits by volatile chemicals. *Science*, **115**, 149–150.

Wenzl, H. (1952). Sclerotinia-Spitzendürre bei der Zweigweichsel. (*Prunus fruticosa* Pall). *Sydowia, Annales Mycologici. Ser* II **6.** 268–289.

Yarwood, C. E. & Harvey, H. T. (1952). Reduction of cherry decay by washing. *Plant Dis. Reptr*, **36.** 389.

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